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An Address.[1]

THE DEVELOPMENT OF THE STUDY OF THE HISTORY OF MEDICINE.

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My first duty today is to express my appreciation of the honour which has been paid me by the executive of this fifth session of the Australasian Medical Congress in asking me to preside over this Section of Medical Literature and History. An historical section is quite a newcomer at these Australasian congresses; I think that this is only the second occasion on which medical history has been recognized by the authorities. At the congress held in Sydney in 1930 this section was presided over by a distinguished South Australian in the person of the late Dr. A. A. Lendon. Dr. Lendon throughout his life showed great interest not only in Australian medical history, but also in the history of South Australia, and many interesting articles flowed from his pen.

It is my pleasant duty to open our readings and discussions by delivering a presidential address. As one who for thirty years has made a hobby of medical history, I thought I could choose no better subject than "The Development of the Study of the History of Medicine". During these thirty years I have seen the history of medicine, so long the poor relation of medical studies, gradually come into favour, and at the present time it forms a subject in the medical curriculum in many schools in Europe and America.

The beginnings of medical history can be traced back into remote periods, for we find it a well-

[1] President's address, read before the Section of Medical History and Literature at a meeting of the fifth session of the Australasian Medical Congress (British Medical Association) in August, 1937.

established custom among the ancient writers to preface their works with a description of the doctrines and advances made by their predecessors, and in recent times there has been a revival of this useful custom. In our modern text-books many of the articles are prefaced by historical summaries, and this custom is largely due to the teaching of Sir William Osler, who, when lecturing to his students at the bedside, told certain among them to look up the original authorities and to read out to the class the original description of the disease from which the patient was suffering.

If we study the works of the ancient Roman author Celsus we shall find the first attempt at a history of medicine. In the *Proœmium* to his book *"De Medicina"*, Celsus gives us a most complete account of the origin and progress of medicine, describing the different sects into which physicians were divided and telling us of the doctrines which ruled the medical world at the time when Greek culture was at its zenith. Celsus has preserved in his book many facts about the ancient physicians which would have otherwise been lost.

Celsus remains one of the enigmas of medical history. Until quite recently his book, thanks to the elegance of its Latinity, was a favourite text-book, and was used as a test of the knowledge of Latin which was until quite recently required of the medical student. It is generally accepted nowadays that Celsus was not a medical man, but was one of those men who from time to time in the world's history, driven by a thirst for learning, make all knowledge their province. His book on medicine is only one volume of a series which dealt with each branch of science. Unfortunately only the volume on medicine has come down to us.

Thus we may look upon the old Roman patrician Aurelius Cornelius Celsus as our first medical historian.

Fifteen hundred years pass by, years in which, after the death of Galen in A.D. 200, medical science gradually deteriorated and was kept alive only in such refuges of learning as the monasteries. With the revival of learning and culture which followed the discovery of printing, and with the expansion of world knowledge which followed the discovery of America and the voyages to the countries of Asia, medical science underwent a new birth.

We meet our next medical historian in the person of Symphorien Champier, who in 1506 published the first series of medical biographies, entitled "On Famous Writers on Medicine". Champier has an additional interest for medical historians in that at Lyons he became the friend of Michael Servetus, the discoverer of the lesser circulation, whose tragic story has been so graphically told by Osler in one of his essays. Servetus, while living in Lyons, acted as press reader for the Trechsels, a firm of printers who published many of Champier's books. We read that Champier (or Campeggius, as he latinized his name) was

> a man of large and liberal culture, of a truly noble nature, an admirer of learning and a patron of the learned. Fearless in bringing help on the battlefield, to which he accompanied his chief, the Duke of Lorraine, he was no less ready to brave pestilence in the city and was as often to be seen in the hovels of the poor as in the palaces of the great and wealthy—a true physician, a great and good man.

Champier was physician to Charles III and to Francis I, and was one of the founders of the medical school of Lyons. His books have only a bibliographical interest nowadays, but in his medico-historical researches Champier was a pioneer.

The next two hundred years produced very little of importance in medico-historical studies. Mention may be made of the biographical dictionary of the Flemish doctor Pierre du Chastel or Castellanus, for many years the only book in its field. A few words must be devoted to the enthusiast René Chartier, who was born in 1572. Chartier, occupied as he was with a busy practice, spent the spare moments of his life and 150,000 francs in producing a complete edition of the works of Hippocrates and Galen in thirteen huge folio volumes. He travelled widely in Spain, in Italy and in England, and was physician to the French princesses of his day. He lived to the ripe old age of eighty-two, and is an outstanding example of the truth that hard work does not shorten one's days.

With the publication of Daniel Leclerc's "History of Medicine" at Geneva in 1696, medical history entered upon a new phase. Arnold Klebs tells us that:

> The impetus to study medical history may be traced to the collections of objects of antiquarian interest which were collected by many medical men in the seventeenth century. All this material had to be sifted and studied, and two cities and two medical men share the honour of producing the two first scientific histories of medicine. Geneva and London are the cities, Daniel Leclerc and John Freind the medical men.

Daniel Leclerc was born at Geneva in 1652, and throughout his life took a leading part in the affairs of his native city. His knowledge of Greek and Latin was profound. He was fortunate in having at his command the fine libraries collected by his uncles. He studied medicine at Paris and took his degree at Valence. Leclerc's history can still be consulted with profit, but, as Garrison somewhat unkindly says:

> A wholly uncritical approach, portentous ignorance of or indifference to the basic sources of fact, an overweening assumption of omniscience and, in consequence, a facile tendency to improvisation are outstanding traits of Leclerc. Foreknowledge absolute of the status of medicine before and after the Flood is taken for granted, and even the worries of Noah about the provisioning of the paired animals in his ark and the disposal of their excreta is considered in one instance.

In the eighteenth century there was a remarkable extension of interest in the history of medicine, and this century was to witness the foundation of many medical societies such as the Royal Prussian Academy of Sciences, founded in 1700; the English Royal Society was founded in the previous century.

The medical periodical became a force in the medical world. Garrison tells us that in the eighteenth century the medical periodicals numbered well over one hundred, and among them we have the first medico-historical journal. This was the short-

lived *Archiv für die Geschichte der Arzneykunde*, published by Philipp Ludwig Wittwer in 1790. This journal lasted only two years, dying with its founder.

To Kurt Sprengel we owe the next medico-historical journal, the *Beiträge zur Geschichte der Medicin*, which was also short-lived and was published at Halle in 1794. Of its founder, Kurt Sprengel, I shall have more to say later on.

The founder of the study of the history of medicine in England was Dr. John Freind, who was born in 1675 and was a graduate of Oxford University. He died in 1728, and is honoured by a monument in Westminster Abbey. Freind had one of the largest practices in the England of his time. In 1703 he published his first book "Emmenologia", and lectured on chemistry. He accompanied the Earl of Peterborough to Spain as physician to the army. After the war he travelled in Italy, and on his return wrote a defence of the conduct of his friend and commander the Earl of Peterborough.

In 1722 the government of the day believed that they had discovered a Jacobite plot, and Freind, amongst others, was arrested and imprisoned in the Tower of London. Here our historian remained from March 15 until June 21, 1723. During his stay in prison Freind occupied his time in beginning his "History of Physick from the Time of Galen to the Beginning of the Sixteenth Century". He was prompted to carry out this task by receiving from Dr. Mead, who visited him often, a copy of Leclerc's "History of Medicine", with a request to express his opinion of the work.

Freind's work is a continuation of the work of Leclerc, carrying the story down to the sixteenth century. This book is still interesting to read. Freind's knowledge of Arabic helped him to give by far the best account of the Arabian physicians up to his time. Freind was a man of sound scholarship and a good example of the cultured physician. His friend Richard Mead did more for medical history by his collections and his magnificent library, which is now in the British Museum, than by his writings. The famous Dr. Samuel Johnson was a friend of Mead and remarked of him that "Dr. Mead lived more in the broad sunshine of life than almost any man".

It was largely through the intercession of his friend Mead that Freind was liberated from the Tower. Mead was called to attend Sir Robert Walpole, the Prime Minister of the day, and pleaded Freind's cause extremely well, refusing to prescribe for the patient unless the prisoner was set at liberty. In that delightful book "The Gold-headed Cane", we read:

> The evening after Freind's release, there was a numerous assembly at Mead's house in Great Ormond Street, attracted by the hope of meeting Freind, and congratulating him on his liberation from the Tower. When the party broke up, and Freind and Arbuthnot were about to take their leave together, as they lived in the same part of the town, Dr. Mead begged Freind to step with him for a moment into his private study, which was a small room adjoining the library. There he presented him with the sum of five thousand guineas, which he had received from Freind's patients, whom he had visited during his imprisonment.

No discussion of the medico-historical movement of the eighteenth century would be complete without mention of the herculean bibliographical labours of that Admirable Crichton of knowledge, Albrecht von Haller, who was born in 1708 and died in his seventieth year. Haller was the greatest physiologist of his time, and it is said that his writings contain many discoveries which we have rediscovered. He taught all branches of medicine at Göttingen, wrote 13,000 scientific papers, and was equally eminent as an anatomist, physiologist, and botanist. He wrote poetry and historical novels and founded the science of medical bibliography. Haller most successfully utilized in his practical work the teachings of medical history, the discoveries and errors of his predecessors serving as a guide and control in his own labours. His four great bibliographies, "Botanica", "Medica", "Chirurgica" and "Anatomica", were founded on the books in his own library, and he was the first to index the articles in medical periodicals, utilizing as copyists his lady relatives and their friends, who did the work for pin-money.

Lectures in the history of medicine may be said to date from the last years of the eighteenth century. In many of the universities in Germany lectureships were established, and after the revolution in France a chair in the subject was established at the faculty in Paris. This chair was short-lived, but was revived in 1870 and held for a short time by the historian Charles Daremberg. It is still in existence.

The last quarter of the eighteenth century produced one of the greatest medical historians in the German botanist Kurt Sprengel, who lived from 1766 until well into the nineteenth century, dying in 1833. His history, which runs to six volumes, was translated into French and Italian; it has been a quarry for many later historians. Garrison calls it "a marvel of solid learning". Like all German medical histories, it amazes one by its industry while tiring the reader by its involved style. Sprengel inaugurated what has been called "the epoch of applied research in medical history". The interest of historians of previous epochs had been centred chiefly in classical Greek, Roman and Renaissance medicine; Sprengel arranged it in distinct epochs and schools, or in relation to the various branches of the healing art. He may be said to be the first medical historian who treats the subject from a philosophical point of view.

Sprengel's writings gave birth to a school of medical historians of whom I may just mention Hecker and Haeser. Hecker wrote the first history of epidemics, and his book on the Black Death is most interesting. The most scholarly history of the period was written by Heinrich Haeser, who in 1839 was professor of medicine at Jena. Haeser was the son of a music director at Weimar, and was educated in an atmosphere of culture to become one of the most learned physicians of his time.

In France the ablest historian of medicine was Charles Daremberg, a native of Dijon, who, besides editing various medical classics, wrote what is by many good judges considered to be the best history of medicine in French. Daremberg was a great friend of Emil Littré, who is famous for his work in philology and, above all, for his gigantic five-volume dictionary of the French language. Littré lived for eighty years, and besides his dictionary, produced an edition of the works of Hippocrates in Greek and French in ten volumes.

As the nineteenth century grew to maturity medical historians sprang up in every country, and it is impossible to name them all. Germany, Italy and Spain all produced creditable work. In England authors concentrated on the biographical aspect of the subject—for example, Munk's Roll of the Royal College of Physicians—or wrote monographs on special subjects—for example, the studies of J. F. Payne on Anglo-Saxon medicine and Norman Moore on the history of St. Bartholomew's Hospital.

The middle years of the nineteenth century were lean years for medico-historical studies. It has been said that the great advances of medical science, such as the discovery of anæsthesia, the discoveries of bacteria by Pasteur and Koch, and the great advances of surgery resulting from Listerism, so occupied men's minds that they had no time to think of the past, and a school of thought grew up that regarded historical research in medicine as waste of time.

With the advent of the twentieth century, historical medicine, following in the footsteps of general history, archæology and medical science in general, has made great progress. Scientifically trained investigators have increased in numbers and have made use of improved instruments and methods of research. Ancient texts have been reedited and the work of the excavator has thrown much light on the origins of the medical art. The anthropologist has also lent his aid, especially in describing the medical practices of primitive peoples.

The nineteenth century witnessed the establishment of several medico-historical institutes. The first was founded at Leipzig, thanks to a benefaction left by Theodor Puschmann, who was professor of the history of medicine in Vienna. He and his wife were comparatively wealthy, and the University of Leipzig received the sum of 500,000 marks. To control this institute came the greatest of all medical historians, Karl Sudhoff, and by his influence and teaching the study of medical history has been revolutionized.

Sudhoff was the son of a theologian and philosopher of Luxemburg, and was born at Frankfort-on-Main in 1853. For thirty years Sudhoff conducted a large practice at Dusseldorf. His early studies had been philological, and his interest in the Middle Ages caused him to study mediæval medicine; from this he passed on to study the works of Paracelsus. His studies on Paracelsus have resulted in a complete revaluation of the work and writings of this famous physician, who had for many a year been held up to scorn as a quack and charlatan. It is now realized that medicine owes much to the iconoclastic zeal of the Swiss physician, and that behind his bombastic exterior and loud boasting there was a great personality. With the foundation of the Puschmann Institute at Leipzig in 1901 the chair and directorship were offered to Sudhoff. After some hesitation he accepted, and from then on Sudhoff travelled all over Europe, copying manuscripts and documents, seeking in the libraries and monasteries for the original sources of medical history. He established in 1907 his *Archiv für Geschichte der Medizin*, which became and still is the most scientific journal devoted to this study. It would be utterly impossible to give you any idea of the vast amount of pioneer historical work accomplished by Sudhoff. Monograph after monograph came from his pen, and in addition he trained many pupils, one of whom, Henry Sigerist, has proved as great a scholar as his master, and is now in charge of the Institute of Medical History at Baltimore. Of him more will be said later.

Meanwhile medical bibliography had become an exact science. Following on the heroic labours of Haller in the eighteenth century, the German Ludwig Choulant had compiled many works which are still invaluable. But it is to the United States that we owe the greatest medical bibliography of all time. I refer to the Index Catalogue of the Surgeon General's Library. This work is due mostly to the efforts of John Shaw Billings and Robert Fletcher. The catalogue embraces the contents of a library containing over 800,000 items, and is of inestimable value to the medical man who is hunting up the authorities on any subject. The work of Billings was carried on by another great medical historian, Fielding Garrison, who in 1913 published his "Introduction to the History of Medicine". This book has proved a great boon to all those interested in medical history, and has already passed through four editions. It is far and away the best reference book, and is a mine of information. Garrison, a great lover of music, wrote many interesting historical studies and died only in 1935. A man of retiring disposition, he was always ready to help those who were in difficulty with their historical studies, and he carried on a correspondence with medical men all over the world.

In England the interest in our subject has grown more slowly. The International Congress of Medicine in 1913 formed a section for medical history under the presidency of Sir Norman Moore, and from this beginning arose the International Congresses of the History of Medicine, of which ten have now been held. The last was held in Madrid in 1935, and it is sad when one reads of the pleasant happenings at that congress to realize that many of the beautiful buildings and the treasures of the past which were displayed with so much pride by our Spanish colleagues, have been destroyed.

During the last few years lectureships have been founded at Edinburgh by Comrie and at University College, London. At the latter, lectures have been

given by Charles Singer, who, apart from his delightful "History of Medicine", has written the best history of biology. The Royal Society of Medicine has a section which has flourished greatly under the leadership first of Osler, then of Henry Morris, Norman Moore, D'Arcy Power and many other distinguished men. Charles Singer and his wife have done much pioneer work on the mediæval manuscripts. Throughout England and Scotland the interest is growing and the quality of the work produced has greatly improved.

As regards the teaching of the subject, throughout Europe we find chairs and lectureships springing up on all sides. In Leningrad the Soviet has established an "Institute for the Study of the History of Science", including medicine. In Poland and Roumania, in Italy, and even in Turkey the subject is arousing a live interest.

In Baltimore in the United States of America an institute under the directorship of Henry Sigerist has been founded at the Johns Hopkins University, and from this institute there issues a monthly bulletin. At this institute there is a permanent staff, the members of which, apart from their teaching work, are engaged in full-time research and are busy in reediting the ancient classics. Here are to be found the most up-to-date methods of teaching medical history to students. The instruction extends over the four years of the medical curriculum. A short lecture course is offered to the freshmen class in the beginning of the academic year, which is a mere introduction tracing the broad lines of development, the history of the leading ideas and the leading men, and acquainting the students with the classics of medical literature. The essential instruction then is given in seminar courses, the subjects of which are related in one way or another with the curriculum of the year, and in which the students take an active part. An interesting innovation is the preparation by the students of exhibits illustrating various phases of medical history. Two students take a subject, for example the history of anatomy, and make a selection of pictures and books illustrating their special field; they then read a paper on the subject, which is discussed by the class as a whole.

Journals devoted to medical history have sprung up on every side, published in every European language. The most comely of these is the American one published by P. B. Hoeber of New York, "The Annals of Medical History".

What have we done in Australia to advance the study and the knowledge of medical history? Up to the present time very little. I am not forgetful of the doctors who have delved into the records of our past, men like Dunlop, Lane Mullins and Lendon, who have left us, nor of the good work done by men who are still with us; but so far there has not appeared a medical man to write the complete history of the profession in Australia. I think that the time has come when such a task should be undertaken. It might be difficult for any one man to find time to deal with every State. Why not divide the task, each State being given to someone versed in the medical history of that State? When we have the histories of the various States completed they could be edited in one volume, and we should have at last a history of the medical profession in Australia.

As to the teaching of general medical history in our medical schools in Australia, there is very little to record. In Sydney more than thirty years ago the late Dr. Fiaschi gave a few lectures, but the course soon died. In Adelaide Dr. Bernard Dawson has given a course of lectures to junior students and has written an excellent synopsis of medical history which forms a splendid text-book for the student. For the last six years in Sydney I have given a course of twelve lectures and bibliographical demonstrations to sixth-year students. The attendance is voluntary, and averages 70% of the students.

I fully realize that the medical curriculum is already overweighted, and that to add any more burdens to the medical student would be sheer cruelty; but I do think that a short course of at least twelve lectures on medical history should be given in all our medical schools. No examination need be held, and attendance, while not compulsory, should be encouraged; and, judging by our experience at the medical school of the University of Sydney, students welcome these lectures. As far as possible the lectures should be illustrated by lantern slides and by the exhibition of books. This course might help to instil a little general culture into the average Australian practitioner, who at present, owing to the absence of proper library facilities, is in danger of becoming merely a skilled artisan and of lacking any knowledge of the great traditions of the learned profession to which he belongs.

I know that the proper teaching of medical history requires time as well as properly endowed and equipped libraries, and I say without fear of contradiction that the medical libraries in Australia are no credit to the medical profession. Once again I will reiterate what I have often said before: we must build up worthy medical libraries in Australia if we are to keep in touch with medical progress in other parts of the world. When one views the great medical libraries of American cities and schools half the size of Adelaide, Melbourne and Sydney, which have been endowed by patriotic citizens, one is filled with envy. One wonders why in Australia such patriotism is so seldom seen. Cannot we copy the example of Osler, who influenced many of his wealthy patients to establish libraries?

I have taken up much of your time this morning and we have much to do, but let me close by quoting the words of a leading American medical scholar, Dr. Eugene Cordell, of Baltimore, who sums, as follows, the advantages of the study of the history of medicine:

1. It teaches what and how to investigate.
2. It is the best antidote we know against egotism, error and despondency.
3. It increases knowledge, gratifies natural and laudable curiosity, broadens the view and strengthens the judgement.

4. It is a rich mine, from which may be brought to light many neglected or overlooked discoveries of value.

5. It furnishes the stimulus of high ideals which we poor, weak mortals need to have ever before us; it teaches our students to venerate what is good, to cherish our best traditions, and strengthens the common bond of the profession.

6. It is the fulfilment of a duty—that of cherishing the memories, the virtues, the achievements of a class which has benefited the world as no other has, and of which we may feel proud that we are members.

THE RETINA OF THE AUSTRALIAN MAMMAL.[1]

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THE retina of all vertebrate animals is constructed on the same general plan, and with few exceptions may be divided into the ten layers familiar to the student of the human eye. Variations in the structure of the different layers occur from group to group, from species to species, and even amongst the individual members of a species. Whilst the main variations depend on the taxonomic position of the animal, the finer variations would appear to depend on its habits. The avian retina is characterized by the fact that the outer nuclear layer contains only two rows of nuclei, whilst the ganglion cell layer is much thicker, a condition the reverse of that occurring in any vertebrate retina except the sauropsidian. The ratio of rods to cones will depend on the habits of the animal, whether they are diurnal, nocturnal or crepuscular, as will also, apparently, the presence or absence of colour in the oil droplets.

This paper is concerned mainly with the structure of the rods and cones of the Australian monotremes and marsupials. Although investigations made years ago on the kangaroo (*Macropus giganteus*),[1] wallaby (*Wallabia rufogrisea bennetti*)[1] and platypus (*Ornithorhynchus*)[2] revealed features in the cones which presented a marked difference from their higher mammalian relatives, no further observations have been published on this interesting subject. Furthermore, doubt has been cast on the findings of the earlier investigators owing to difficulties of technique unavoidable at the time.[3] When one considers that Marcus Gunn[2] made his observations on the eye of the platypus from a specimen presented to him by "a gentleman at Ealing" and which had been preserved in whisky (or as an American writer[3] naïvely puts it, "*ca.* fifty per cent alcohol"), his description is amazingly accurate.

With few exceptions every vertebrate retina contains two kinds of visual cells, the rods and cones, the latter functioning in photopic vision, the former in scotopic. The distinction cannot always be made on anatomical features alone, and Walls[4] has recently brought forward evidence to prove that a structure which is anatomically a cone functions in some species as a rod. Whilst there appears to be no doubt of the truth of the duplicity theory of vision, many factors must be taken into consideration before a particular cell is ultimately classified as a rod or cone. The following table constructed by Walls contains most of the features necessary to differentiate a rod from a cone.[5]

Rods.	*Cones.*
Connected in multiple to bipolar neurones.	Connected singly, or nearly so, to bipolars.
Outer segment of maximal volume in order to carry on maximum photochemical activity, and therefore cylindrical.	Outer segment of minimal volume, and thus conical unless inner segment is filamentous, as in Muridæ.
Contain visual purple when dark-adapted.	Never contain visual purple.
Elongate in light and contract in darkness, if they move at all.	Elongate in darkness and contract in light, if they move at all.
Predominate in nocturnal animals, sometimes to the exclusion of cones.	Predominate in diurnal animals, sometimes to the exclusion of rods.
Often have a compact end-knob devoid of lateral branches.	Always have a dendritic end-knob.
Never contain coloured oil droplets.	Often contain coloured oil droplets.
Never doubled unless secondarily formed from double cones (geckos and two or three snakes).	Double or twin cones in all but lampreys and mammals.
Nuclear chromatin often in large pieces without linin.	Nuclear chromatin always in small pieces, on linin network.
Usually have the nucleus below the limitans.	Usually have the nucleus in contact with the limitans.

Double or twin cones consist of two individual elements fused in a greater or less part of their length. The sensitive end piece, as well as the dendritic foot piece, is always separate, and the two elements probably function as two independent units. In twin cones, which occur in the fishes and, as I have shown,[6] in the marsupial, the members are similar in structure to each other as well as to the single cones. The units of a double cone are quite unlike each other and are referred to as the principal and the accessory cones. The principal has the larger nucleus, contains an oil droplet if they happen to be present in the retina, and resembles in every way the single cone. The nucleus of the accessory is often much smaller, there is no oil droplet present, although the oil is said to be finely scattered throughout the ellipsoid, and the myoid often contains a paraboloid—a delicate globular structure. The oil droplet is in the distal extremity of the ellipsoid, and the light rays must pass through it before reaching the outer member. Until the discovery of oil droplets in the cones of marsupials by Hoffman[1] and of *Ornithorhynchus* by Gunn,[2] it was thought that they were not present in the retina of the mammal. Greef[7] and Rochon-Duvigneaud[8] state that double or twin cones are not present in mammals. Gunn does not mention them in his article. I have not been able to obtain Hoffman's description, but it is safe to assume from the statements of both Greef and

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

Rochon-Duvigneaud that he did not observe double or twin cones in the kangaroo or wallaby.

Some two years ago Professor Wood Jones suggested that I might use some material that he had collected for the investigation of the eyes of the Australian mammals. A study of the retina of the ring-tail opossum (*Pseudocheirus laniginosus*) and of the native cat (*Dasyurus viverrinus*) confirmed the findings of Hoffman as regards the presence of oil droplets in the cones.[6] A further finding was that of the presence of twin cones each with an oil droplet. These structures are quite different from the sauropsidian double cone, and to find similar structures, that is twin cones with oil droplets, it is necessary to go back to the sturgeon. Unfortunately, fresh material, which is so essential for these studies, is very difficult to obtain, and it will probably be a long time before an extensive study of the retina of the marsupials is completed. Examination of another opossum (*Trichosurus vulpecula*) revealed the presence of oil droplets and twin cones. When the fresh retina was examined with apochromatic lenses the oil droplets were seen to be colourless. Examination of the retina of a kangaroo (*Macropus giganteus*) which had been dead about twelve hours, again showed colourless droplets. This is at variance with the findings of Hoffman (as reported by Rochon-Duvigneaud), who states that they are blue, green and red.

Examination of the retina of the platypus confirms the findings of Gunn as regards the presence of oil droplets. I have not yet been able to examine a fresh retina to ascertain whether the droplets are coloured. Double cones are present and are of quite a different structure from the twin cones of the marsupial. The chief member contains an oil droplet, whilst the accessory cone in close apposition with the other has none. In every case, as far as I could see, the nucleus of the accessory cone was ectopic, that is, it was outside the external limiting membrane. This may depend on the state of light adaptation of the animal, as it is well known that with elongation of the myoid the nucleus tends to pass through the limitans. This can be seen in the sections of the eye of the trout, and is not uncommon in sections of the human retina. In the sections of the platypus one is at first apt to think that the ectopic nucleus is a paraboloid. Closer examination reveals its true nature. The presence of the ectopic nucleus makes the identification of double cones easy, although I am not at all sure that similar structures are not present with the nucleus inside the limitans. The visual cells are very small, and even with oil immersion lenses the identification of detail is not easy. Single cones are much more numerous than double, and rods and cones are present in approximately equal numbers. The rods differ from those of the marsupial in being less numerous and much more massive.

The remarkable feature of the visual cells of the platypus is their resemblance to those of the sauropsidian and not to those of the marsupial or higher mammal. This is obvious on comparison with sections of the retina of the goanna (*Varanus varius*) and the pigeon (*Columba livia*). The outer nuclear layer differs, however, in containing four or five layers instead of two. Although there is no great reduction in numbers as regards the nuclei of the cells in the inner nuclear layer as compared with the same layer in the opossum and native cat, it is obvious from the thinness of the layer of nerve fibres that there is a great deal of summation present. When one considers that in addition to bipolars there are present in the inner nuclear layer amacrine, horizontal and nuclei of the Mullerian fibres, it is evident that a great part of the summation has taken place as usual, between the inner and outer nuclear layers.

A further point of resemblance to the sauropsidian eye is in the cartilaginous sclera present in both *Ornithorhynchus* and *Echidna*. The anterior bony circlet is missing. Unfortunately, the only specimen of *Echidna* which I have been able to obtain had been dead for some hours before it was possible to fix the eyes, and the precious outer layer had disintegrated.

The difference between the visual cells of the monotreme, marsupial and higher mammal provides another interesting problem in the study of the comparative anatomy of the eye. It is impossible to say what is the function of the double or twin cone. The double cone reaches its highest development in the eye of the bird, an organ which probably excels that of the mammal in its efficiency as an optical instrument. Many theories have been wrapped round the oil droplets, including theories on colour vision. Whilst they are coloured in round-pupilled diurnal forms, they are colourless in the slit-pupilled nocturnal forms. *Trichosurus vulpecula* is nocturnal in habit, and its pupil is a small, vertical oval even in daylight of an intensity of much less than a hundred foot-candles. *Dasyurus viverrinus*, which is at least crepuscular in its habits, possesses a pupil which contracts to a similar size only in brilliant sunshine. One fascinating theory gives the coloured droplets the function of colour filters to sharpen vision.[3] In the higher mammals their function, according to this theory, is assumed by pigment in the lens, by the yellow colour of the thin layer of blood in the retinal circulation, and by the yellow pigment at the macula.

The retinal circulation of the monotremes and marsupials resembles that of the different species of the higher mammals. There is no pecten or cone, only in some instances a network on the disk, which Lindsay Johnson has depicted in the kangaroo, wallaby and bandicoot.[9] The retina of the platypus and *Echidna* is avascular, as are the majority of the marsupials which have been observed. The Dasyuridæ, as well as the Virginian opossum (*Didelphys*) have a well-developed retinal vascular system. In the sections of *Dasyurus viverrinus* the capillaries extend, as in the human, to the inner half of the outer molecular layer.

Lindsay Johnson pictures the Tasmanian devil (*Sarcophilus harrisii*) as having a well-developed central retinal vascular system. There is, however, as great a variety of retinal and chorioidal structure among the marsupials as the higher mammals. (Whilst Lindsay Johnson was unable to find a tapetum in any of the marsupials he examined, *Dasyurus viverrinus* has a well-marked fibrous one.) It is difficult to understand why such great differences should exist. In addition it is interesting to speculate as to the exact function or functions of the retinal circulation. The bird, with its cellular retina and keen vision, gets on very well without it, and in man that part which has the keenest vision, the macula, is avascular. Like many other features in the comparative anatomy of the eye, it still presents an unsolved problem.

Acknowledgements.

In conclusion, I should like to thank Professor Wood Jones for the material he has placed at my disposal and for the manner in which he has done everything possible to facilitate the work, Sir James Barrett and Dr. Edward Ryan for further material and help with the literature, and Mr. H. Marriott for his assistance in preparing specimens and photographs.

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SOME RESULTS ACHIEVED BY TUBERCULIN THERAPY.[1]

By L. ELWELL,
Brisbane.

A STATISTICAL comparison of methods of treatment of pulmonary tuberculosis is notoriously fallacious unless based on the period of survival of the patient. For the assessment of my results in the treatment of pulmonary tuberculosis with tuberculin I have taken all those cases in which treatment commenced at least ten years ago and was continued for a minimal period of six months and mostly for more than twelve months. There are 93 cases in all. Sixty-three of the patients were returned soldiers, all accepted by the Repatriation Commission as suffering from pulmonary tuberculosis. Of the remaining thirty, twenty were females. In the big majority of these thirty cases tubercle bacilli were found in the sputum; in the few remaining cases, in which sputum was either absent or contained no discoverable tubercle bacilli, a positive diagnosis was made by radiography and/or the subcutaneous tuberculin test in conjunction with physical signs and symptoms. The large majority were going down hill in spite of rest and every climatic advantage, as evidenced by rapid and progressive loss of weight and increase of symptoms; the remainder were making no headway. According to the Turban-Gerhardt classification, none of these patients was in Stage I. Of twelve patients in Stage II when treatment was commenced, ten are alive and have been well for twelve to eighteen years. The remaining two may be alive, but cannot be traced. If these are counted as dead the percentage would be eighty-three.

Of the 81 patients in Stage III, 43 are still alive. If the three who could not be traced are counted as dead, 53% are still alive, their average survival period being sixteen years, and 68% survived for ten years. As available mortality statistics do not allow for a follow-up of longer than ten years, this is the longest survival period that is available for comparison.

The importance of utilizing an adequate survival period as a gauge of success is shown by the results Wingfield gives of Frimley, the sanatorium for Brompton Hospital. Here a certain selection of cases is made, inasmuch as only those patients are admitted who have a reasonable chance of being discharged fit for work. The immediate results are that 80% of patients are discharged fit for work. However, at the end of ten years, of patients in Stage II, 45%, as against 83% or more of my small group, were alive, and of those in Stage III, 10% only as against 68% of my tuberculin treated patients were alive.

All the thirty-five deaths of which I have record were of patients in Stage III when first treated. Two were due to suicide, and in each case the patient, a returned soldier, was augmenting his income by manual labour.

A taxi-driver remained very fit and well for thirteen years and then died of pneumonia. A soldier worked his orchard for many years and then died of general paralysis. A woman recently died at the age of seventy-one years, after a very active life during the fourteen years following her treatment. A patient with a laryngeal complication, classed as severe by the laryngologist, after his treatment elected to play competitive football, with the result that one would expect.

A gold-miner, aged fifty years, with tuberculosis superimposed on extensive silicosis, was sent down by his

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

physician from the north to die; he returned home after two years' treatment, and three years later had increased nearly two stone in weight and was reported by the same physician as being remarkably fit; he lived to be fifty-nine years of age.

A professional man, with half his epiglottis gone, suffering from constant dysphagia and dyspnœa on the least exertion, with practically the whole of one lung and two-thirds of the other involved (all of recent spread), survived for nine years and practised his profession for more than half that time.

A mercer, with extensive involvement of both lungs, and with severe tuberculosis of the tarsus on which rest in plaster had made no impression, discarded his caliper and climbed a mountain two months from the commencement of treatment. He carried on his business successfully for ten years.

Many of the others who have since died showed similar striking improvement, even such as to encourage them to run the risk of severe over-exertion. Of the forty-three survivors in Stage III, five had severe urogenital tuberculosis at the time of treatment.

One had a testis removed two years before and reported with a discharging sinus and epididymitis in the other. The testicular condition cleared up promptly with tuberculin, and the patient reported that he was well after eighteen years and able to continue regular light work. Only one of the others had ultimately to have a kidney removed, and he has improved since.

One Stage III case deserves special mention. The patient was sent home from France because he had suffered from fits of unconsciousness in the line. These gradually became more frequent and severe, being accompanied by fever and convulsions and biting of the tongue and lips. He also had constant cough and recurrent hæmoptysis. Before treatment he had lost 9·5 kilograms (a stone and a half) in weight and had had as many as twelve or thirteen fits a day. With treatment the fits promptly began to lessen in frequency and severity; he had two more major bouts only, and for the past twelve years he has had only a very occasional minor seizure. Farquhar Buzzard has described similar cases as chronic tuberculous meningitis.

The virtually complete disappearance of a large goitre, associated with tachycardia and pronounced exophthalmos, occurred without any period of rest or other treatment in a girl, aged twenty-one years, during a six months' course of tuberculin treatment for recurrent pleurisy. She remained very well and quite free from her accustomed colds till she became afflicted with pneumonia and pleurisy six years later; this was followed by the appearance of tubercle bacilli in the sputum. She was given another course of tuberculin, and in spite of continuous office employment has never looked back in the eleven years since.

I have never observed any clinically recognizable effusion during tuberculin administration, and it is interesting to note that Neumann, of Vienna, who uses tuberculin in conjunction with artificial pneumothorax, observes effusion in only 2% of such cases as against the normal 70% to 80%.

I find, however, that artificial pneumothorax is very rarely indicated if the best use is made of tuberculin, and in most of the cases under review artificial pneumothorax would have been out of the question owing to the extensive and fibrotic nature of the pulmonary lesions. Burrell has published results of all cases in which he had induced or attempted to induce artificial pneumothorax at least ten years previously, showing that even in those cases in which less than one-third of the better lung was involved, only 18% of patients survived for ten years, whereas in those cases in which more than one-third of the better lung was involved, not one out of thirty-six survived more than seven years, and all but two were dead within two years. Of his patients with unilateral disease 50% were alive after ten years. Of my patients nine only had unilateral disease, and all but one survived ten years, and that one lived for nine and a half years, representing an almost 100% survival rate. Among my patients with bilateral disease the survival rate for ten years was 69%, whether less or more than one-third of the better lung was involved.

I should like to stress at this juncture that these results could have been far better if it had been practicable to follow the cases up and carry the tuberculin treatment out thoroughly in every case. It is the attainment of the full dose that counts, and again that full dose may need to be reached repeatedly through several courses should there be any tendency to relapse in subsequent years. This is emphasized by the fact that only 52% of those patients who died had a full course, whereas 76% of 43 in Stage III who are living had at least one full course and ten had two or three such courses.

In tuberculous eye conditions, notably irido-cyclitis, Wilkinson has published most successful results in forty-three cases which were referred to him by ophthalmic surgeons for diagnosis and treatment by tuberculin.

I have recently used tuberculin in two cases of Addison's disease with most gratifying results, and I hope that a period of further observation will demonstrate that the striking results already achieved will be lasting.

Reverting to pulmonary tuberculosis, J. R. Gillespie, who presided over the tuberculosis section of the annual meeting of the British Medical Association, has published comparative results which deserve wide publicity. He compares the number of dispensary patients with tubercle bacilli in the sputum at work who had had at least three months' tuberculin treatment with those who had had routine sanatorium treatment only without any tuberculin. In Stages I and II combined, at the end of thirteen years 25% of the tuberculin-treated patients are at work, compared with 8% of those treated by sanatorium methods.

In conclusion, may I submit a plea for the fuller recognition of tuberculin as our greatest asset in the treatment of tuberculosis. Misunderstanding concerning the value of tuberculin has occurred through misapplication, and misapplication can be checked only by the provision of expert instruction in our schools, and in centres such as tuberculosis dispensaries where practitioners should be given the opportunities of studying the treatment as applied in practice by those who know best how to use it. Expert training is just as necessary in the use of tuberculin as it is in the use of the surgeon's knife.

TENNIS ELBOW.[1]

By E. F. WEST,
Adelaide.

DESPITE the extensive literature which has accumulated on the subject of tennis elbow, considerable difference of opinion still exists as to the exact pathology concerned, and as to what constitutes the most satisfactory method of treatment; and it is hoped by this discussion to bring out the opinions of the members on both these aspects, and more especially that of treatment.

First, I shall say very little about symptoms, signs and course of the syndrome. As regards the symptoms, two important facts stand out. First, the typical pain is brought on when a certain movement is executed. In tennis this is usually the backhand shot, more particularly a backhand top spin drive, and players who use this shot are particularly prone to the affection. This shot involves violent and sudden contraction of the radial extensors.

Secondly, when comparatively light objects are lifted in a certain position of the wrist, a sudden inhibition may lead to loss of power, which makes the patient drop the object; the position is that of pronation of the forearm and extension of the wrist. In association with these two symptoms is Mills's test, which consists in putting the fingers and wrist in full flexion and pronation and then extending the elbow. This produces the typical pain felt in the region of the external epicondyle radiating down the back of the forearm to the wrist.

Of the objective signs, the important one is the site of tenderness. In my own experience I have found this to be over the anterior aspect of the external epicondyle, extending down to the level of the joint line; but the maximum tenderness is definitely over the bone. As regards the course, the condition usually clears up in about nine months without any treatment except, possibly, avoidance of the particular movement which causes the pain. In some cases, however, it lasts much longer and appears to defy treatment.

Concerning the mode of production, the onset is not usually acute; the condition comes on gradually after a few days or so of strenuous play. A typical tennis elbow can, however, be produced by direct injury. One patient of mine, suffering from a typical tennis elbow, dated its onset from six months previously, when she had jammed her right elbow in the door of a motor car and, as she put it, "bruised the bone", that is, the external epicondyle.

Ætiology.

We meet with an extraordinary variety of opinions concerning the ætiology. Cyriax[1] in a recent article, which included a comprehensive survey of the literature, collected twenty-six different opinions on the pathology. The main opinions may be stated as follows.

1. An old view is that tennis elbow is caused by strain or tear of the *pronator radii teres*. This can be ruled out, as the pain and tenderness are all on the outer side of the elbow joint.

2. Tennis elbow may result from nipping of the synovial fringes in the radio-humeral or radio-ulnar joint, leading to an internal derangement of the elbow joint. With this view we may place traumatic synovitis of the elbow joint. It was strongly held by Trethowan,[2] who stated:

> From my operative experience I confidently advance the opinion that "tennis elbow" is always a traumatic synovitis of the radio-humeral joint, the trauma being chronic from repeated slight injuries and the failure of the patient to rest the elbow in the early stages, and due especially to excessive extension of the elbow joint and supination of the forearm . . . There is no X ray evidence whatever to support the theory of periostitis, and the suggestion of chronic muscle strain is negatived by the non-occurrence of similar conditions in association with other joints. The pain felt in the elbow and forearm on tightly gripping something with the fingers . . . is explained as being due in part to the increase in pressure on, or constriction of, the synovial pouch protrusion in front of the epicondyle, and in part to the increase of interosseous pressure on the inflamed radio-humeral joint and the nipping of congested synovial fringes.

With all due deference to this authority, I suggest that the explanation is not correct. X ray evidence of periostitis is found in a proportion of well-established cases, and his explanations of the cause of pain on gripping as being due to pressure on the synovial membrane of the elbow joint by overlying muscles is unconvincing.

3. Radio-humeral bursitis may cause tennis elbow. In 1922 Osgood[3] published his findings on the radio-humeral bursæ with reports of three patients (one personal) operated upon. This bursa, lying beneath the conjoined origin of the extensors, undoubtedly exists in a proportion of subjects, as anyone can prove by dissection; evidence that it is concerned in the ætiology of tennis elbow, however, is lacking.

4. Tennis elbow may be due to strain and tearing of the *supinator brevis*. Heald, in his recent book, considers the lesion to be located in this muscle. This will not, however, explain the pain on gripping and the inhibition of certain purely wrist movements. With this theory is associated neuritis of the posterior interosseous nerve.

5. The condition may be caused by tearing of the tendon of the extensor origin from the external epicondyle, with resulting periostitis. This is the view of the ætiology held by the majority, and in my opinion it is the correct one. As previously mentioned, the movement producing the pain is associated with sudden and violent contraction of the radial extensors. The *extensor carpi radialis brevior* arises in common with the *extensor communis digitorum* and the *extensor carpi ulnaris* by tendon from the anterior aspect of the external epicondyle. The short radial extensor is the most laterally placed, and if the point of maximum

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

tenderness is accurately defined, it is found to be the most lateral part of the front of the external epicondyle. Repeated strains of this portion of the tendon cause it partially to tear away from its connexions with the periosteum. Owing to the frequently repeated trauma, healing is unable to take place and a traumatic periostitis is set up. As mentioned before, periosteal bone proliferation is shown by X ray examination in some of the well-established cases. Mills's test places the radial extensor on the stretch and causes the typical pain.

Treatment.

Treatment is not always satisfactory. The cases in which the condition has existed for some weeks or months are the most definite, and in them a very definite response to Mills's test is elicited. Probably most patients can be cured by manipulation under a general anæsthetic. I think general anæsthesia is necessary, and many of the failures occur through this not being employed. The manipulation used is that described originally by Mills[5]—full flexion of the wrist with full pronation of the forearm, the elbow then being brought into full extension. One manipulation is usually sufficient.

Cyriax describes a method of treatment which has been very successful in his hands and which obviates the necessity for an anæsthetic.

> While the patient sits with the elbow held at a right angle and the forearm supinated, deep friction is applied to the anterior part of the lateral epicondyle; the patient's skin moves with the masseur's finger, so as to rub the superficial tissues on the deep structures. This is continued for five to ten minutes—the more chronic the condition, the longer is the period of friction. The elbow is then mobilized . . . The elbow is as fully extended and the forearm is as fully supinated as possible. With one hand on the inner side of the elbow and the other on the outer side of the wrist, the masseur adducts the forearm on the arm with a sharp jerk towards a position of *cubitus varus*. The deep massage to the tender area and the mobilization are repeated thrice weekly until the patient is well; one treatment is seldom enough. In the writer's series an average of four treatments was enough to give complete relief, with extremes of one and nine.

In the only case in which I have tried this method it was successful. It has the advantage of obviating an anæsthetic; its disadvantage is the extra time taken, which is of little moment. In the more recent case the patient usually declines an anæsthetic, and, as has been stated, manipulation without anæsthesia is often unsatisfactory; this is especially so in the recent case, when the patient is apt to resist strongly. Strapping gives relief, but takes a long time to cure. I thing the best method of strapping is tight encircling strapping around the upper third of the forearm. This restricts the contraction of the radial extensors. The patient is able to carry on his tennis more comfortably with this strapping in position. I have not found very satisfactory the pad with a hole cut in the centre over the external epicondyle, because the strapping required to hold it in position cuts into the antecubital fossa and becomes very uncomfortable. Giving up playing tennis for the remainder of the season usually allows the condition to clear up slowly, but is the type of advice not usually appreciated by the patient.

Finally, there are the apparently severe cases which do not respond to manipulation, or in which this is refused. These patients are usually women suspected of being of the neurotic type. In these cases I think the only thing to do is to put the radial extensors at rest by ordering the uninterrupted wearing of a short cock-up splint for about one month.

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IS THE HOSPITAL TREATMENT OF SCARLET FEVER WORTH WHILE?[1]

By F. V. SCHOLES,
Melbourne.

LATER in the course of this paper we shall have to consider exactly what we mean when we speak of scarlet fever. For a beginning we must regard it simply as a notifiable illness, which for generations has been accepted as needing strict isolation, careful nursing and, in the great majority of cases, hospital treatment, the necessity for which arose from its appalling mortality in all civilized countries during the nineteenth century. Early in the present century the continued decline in both mortality and fatality began to arouse doubts as to the need for large scale, almost universal, hospital treatment, and as the incidence of the disease remained high, it became a question as to whether public money should be spent on isolation which was having no effect on prevalence. Since then much has been written and said on the relative methods of hospital and home isolation, and of hospital and home treatment. Up to now both medical practitioners and public have expressed their verdict in no uncertain way; and, dealing as I have done with a very large number of both classes over a period of nearly thirty years, I can see no sign and no likelihood of any change.

After all, the public consists of individuals, and while a citizen may be impressed in an abstract way with the contention that isolation has not reduced prevalence in a particular country or community, he will be certain, and rightly so, that his children in a house or boarding school will be more likely to escape infection if a case of scarlet fever occurring therein is treated by prompt removal of the patient

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

to a hospital. Again, and rightly, most private practitioners realize that as a rule it is dangerous for them to treat cases of scarlet fever in homes and at the same time be engaged in maternity, surgical or even medical practice.

In comparing the well-being of the patients under hospital and home conditions, one has to remember that patients treated at home come under a different heading, social and financial, from that of the average hospital patient. But I have formed the opinion that even under the best conditions of medical attention and nursing in well-to-do homes, the results of treatment of the early stages of the disease are not so good as they are in hospital. If patients could be discharged without fear of consequences as soon as they are well over the acute illness, as is the case with pneumonia, there would, to my mind, be no room for argument as to the advantages of hospital treatment.

Other factors have contributed to the attitude of the public. The costs of treating scarlet fever patients in hospital are borne by the public. As a rule they are met out of municipal and State revenue. It is not for me to say whether the increases in rates and taxes are passed on wholly to the tenant or employee respectively, though one may suspect that they are; or that the wealthy suburb receives a sufficient compensatory protection in return for the high cost of its assistance to the poorer suburb. The fact is that the working man correctly argues that it is cheaper for him to pay his share towards hospital upkeep than for him to have his children nursed and treated at home. With increasing difficulty in obtaining domestic help, and with nursing and medical costs, a working man cannot afford home treatment, especially with its added risk of other cases occurring in the household. Also, with the increase of flat life, shared houses *et cetera*, the proportion of homes in which good treatment can be carried out is diminishing.

It has always been held that severe and complicated cases, and those occurring in destitute or overcrowded homes, should be treated in hospital; and most people will agree that when other occupants are engaged in supplying food, making clothes *et cetera*, when there are other young children, when the case occurs in an institution, boarding house or school, or a block of flats, or when home treatment would mean loss of work and wages for some other member of the family, removal to hospital is desirable. My own observations lead me to the opinion that the public, rightly or wrongly, has accepted, and will continue to accept, hospital treatment for the very great majority of all cases notified.

I believe that in the past hospital isolation has been on the whole a good thing; there is no completely satisfying evidence one way or the other. I am quite convinced that hospital treatment in the early stages of the illness has been, and is, a good thing; and we have now arrived, I think, at a stage at which it is possible to remove many of the unsatisfactory features in the treatment of convalescence. In the last respect, and in that only, the hospital treatment of scarlet fever hitherto has failed.

To study the causes of this failure, and also to see what an impossible position we have got ourselves into by our definition of scarlet fever, it is necessary to go back to the beginning.

Scarlet fever is caused by a streptococcal toxin. If the infected subject cannot produce enough antitoxin in time to prevent it, the rash follows. If he can produce antitoxin reasonably quickly and in reasonable amount, there will be no rash. By the artificial administration of toxin we can produce the syndrome of scarlet fever. By the use of suitable antitoxin we can prevent the appearance of the rash; we can abort it in its early stages, and we can blanche it locally in its developed stage, provided this has not advanced too far.

The portal of streptococcal entry does not matter. Naturally, the upper respiratory tract is the commonest gateway, but whether it be the puerperal uterus, the burn, the paronychia, the urchin's barked knee, or the operation wound, the development or not of scarlet fever will depend on the toxigenic qualities of the invading streptococcus and the antitoxin qualities of the subject. The rash will appear, or will not appear, as a matter of course.

Now, why is it deemed necessary to notify, isolate and treat all cases of scarlet fever, while the great majority of all cases of infection by hæmolytic streptococci can be ignored, at any rate as regards notification and isolation? Or, to put it another way, why should streptococcal infection be regarded as requiring notification and isolation only when it produces a certain group of symptoms? Scarlet fever is a group of symptoms, and nothing more. It is one of the most beautiful syndromes in the whole domain of medicine, but it is merely the manifestation of certain symptoms in certain subjects, while in others who are equally infectious the syndrome is absent, and they are therefore not suffering from scarlet fever in the strict sense of the term.

There is nothing new in this. Before any of us in this room were born it was known that both in and between epidemics there were many instances of "scarlet fever without a rash". Further, that there were many without a fever, and lastly, that there were many without a sore throat.

Epidemics of sickness characterized by follicular tonsillitis, with or without *otitis media*, by nephritis, and by cervical adenitis, have been investigated fully by many observers. The work and the findings are well known, and there is no need to describe them. What I wish to emphasize is that there is no essential difference between these epidemics and epidemics of scarlet fever. Or, to go a step further, if these epidemics had taken place in ideally selected populations, they would have been epidemics of scarlet fever.

Every one of us has met with cases in which tonsillitis in one member of a household has been

followed by scarlet fever in another, and possibly otitis or nephritis in a third. The same streptococcus has caused all of them, so that the symptoms of scarlet fever are merely a result in a particular individual. The cases in which scarlet fever does not appear are not necessarily less severe, or less infectious. Why, then, should selected cases be the only ones which it is thought proper to treat in isolation hospitals? Simply because at the time notification and isolation came into force, streptococcal infection with a rash was the important and dangerous form; nowadays, it is no more important and no more dangerous than that without a rash. It is granted that the raw surface left by the desquamated enanthem makes the risk of dangerous sepsis greater and infectivity longer, but, on the other hand, nephritis and rheumatism are very apt to follow infection without a rash.

It is ridiculous to select one special group of patients for isolation and hospital treatment to the exclusion of the others. I would suggest that cases of acute tonsillitis and otitis should be investigated bacteriologically just as are cases of scarlet fever and diphtheria; that, if practicable, isolation and treatment should be carried out at home, but that if these cannot be provided in the home, the patients should be received in hospital; that if there is not room for all, they should be given preference over certain cases of scarlet fever which can be adequately managed at home, and that in any case the question of hospital isolation and treatment should not be determined by the presence or absence of an unimportant symptom like a rash on the skin.

The same, of course, applies to notification. If it is of sufficient use to justify its continuance, it should include all cases of infection by streptococci which can produce scarlet fever.

Some streptococci are more toxigenic than others; some more invasive than others; some more erythrogenic than others. All the common types have been found capable of causing scarlet fever, given the proper person. Communities differ as well as bacteria. For instance, Griffith's type 10 (Dochez) occurs in England, but has not been found to cause scarlet fever there. It has caused scarlet fever in Russia and America. Type 14 (Barker) has caused outbreaks of tonsillitis, *otitis media*, and puerperal fever in England, but has not been found in scarlet fever there; it has caused scarlet fever in Jugo-Slavia. It is hardly a speculation to say that surely there is no hæmolytic streptococcus, however feebly toxigenic, that cannot cause scarlet fever in some unfortunate person somewhere.

A population of scarlet fever patients, therefore, brought from different localities over a wide area, and aggregated in a fever hospital, will on admission be found to be infected with differing strains or types of streptococci. According to the type, its toxic and invasive powers, the constitution of the patient, the condition of his nose and throat, and his antitoxic and antibacterial powers, so will the symptoms of the acute stage of the disease be determined. These symptoms can be dealt with by modern methods, and nowhere better than in a hospital. Some patients are overwhelmed and killed; others develop suppurative and ulcerative processes such as otitis, rhinitis and glossitis, or cervical adenitis, abscess or angina; but these effects are not usually preventable, and there need be no reproach. Most patients do well, and enter upon a period of well-being after the subsidence of the acute illness, which after all is the disease, scarlet fever.

All goes well until a new era is entered upon, this beginning most commonly about the end of the third week.

From then on, one never knows what is going to happen. From then on, especially in children under the age of nine or ten, too often there is a sinking into a condition of intermittent (or chronic with exacerbations) tonsillitis, adenitis, and rhinitis, pallor, paronychia, and so on. In others there is a quite acute tonsillitis, tending to recur, or a sudden otitis. In others again, though this occurs also in older children, the development of nephritis or rheumatism takes place. In others, classical relapse occurs, with the reproduction of the whole picture of scarlet fever. In fact, the really unsatisfactory and sometimes the really dangerous period of scarlet fever is after the patient has recovered from the disease, and often after he is out of bed. All young children are not so affected, of course; most of them avoid such troubles, but for certain children the later weeks of "convalescence" are a period of chronic naso-pharyngeal infection, punctuated by any or all of the developments mentioned.

For many years this trouble has darkened my young life, and, going one better than Mary of Calais, I fear that the words "scarlatinal convalescence" will be found engraven on my heart. At the 1929 session of congress in Sydney I read a gloomy paper on the subject, and in 1931 a longer and even more gloomy one before the Victorian Branch of the British Medical Association. Many of us had regarded cross-infection by more virulent or more toxic strains as at any rate a major factor, but though this could be controlled by careful nursing while the patients were in bed, prevention was not possible afterwards, except by the rigid isolation of every child from its fellow. This was, of course, out of the question, not only because of the cost and difficulty, but because in its psychological effects on the child it could be regarded only as inhuman and cruel.

During recent years, however, the pioneering work of Griffith in the classification of serological types, which he has carried on continuously for many years, has borne fruit, and its application by Allison and others in carefully managed experiments has shown definitely that the unsatisfactory state of affairs described is due mainly to reinfection by a second or third strain of streptococcus. I am loath to believe that this is always the case; I have seen nephritis, adenitis, and even relapse occur under conditions in which I have thought it most unlikely that fresh strains could be introduced. However, it is now certain that children are infected with

fresh strains, that complications follow, and that the transmission is usually direct, or nearly direct, from child to child. Other possible modes may be mentioned. Infection may be air-borne. Streptococci do travel in the air of a badly ventilated ward, and not only in the still air. In such wards, of course, the bedspace must be increased and ventilation improved. But in this country, where wards can be freely ventilated, summer and winter, and where doors and windows can be open day and night, I do not consider air-borne infection important. Dosage is a large, probably often a determining factor in infection, and though streptococci from certain patients have been shown by Brown and Allison to come to rest on distant bedside lockers, it does not follow that they are likely to be deposited on mucous membranes in sufficient dosage to cause infection.

To make up for this, here flies may carry infection, especially in young children's wards, where many of the patients have rhinorrhœa. The problem of combining the freest ventilation with complete protection against flies is not so easy as it seems, as it is not always possible to wire all doors and windows. All available measures must be taken to keep a ward free of flies.

Nurses and doctors may convey infection from patient to patient. By carrying out the strict principles of barrier nursing, it will not be transferred by hand, but those who are temporary or permanent carriers of streptococci in the throat or nose constitute a real danger. All sore throats must be reported, unhealthy tonsils *et cetera* must be attended to, and heavy carriers of streptococci must not be permitted to nurse young children with scarlet fever.

Carriage of infection by air, flies, nurses and doctors can be controlled, though perhaps not eliminated, and I think that we are now in a position to control infection from child to child. Of course we have not been idle during the past years. The principles of barrier nursing have been steadily creeping into routine nursing practice. Then, also, by means of smaller and subdivisible wards, we have been able to segregate patients according to clinical type. For example, patients with otitis, adenitis, rhinorrhœa, paronychia, or gross sepsis have been nursed in small wards. But it is possible now for us to go further, to segregate patients according to bacteriological type.

I hope at some future date to describe the working of a complete and satisfactory system in detail, but here shall do no more than sketch in outline what we wish to do.

1. Individual isolation of all patients until typed, either in cubicles, or by barrier nursing in small wards or standard wards. While typing is proceeding, the highest degree of nursing must be maintained.

2. Segregation of patients according to type, in wards of suitable size, according to the number of patients infected by each type. For this purpose, subdivisible wards, of which we have a number at Fairfield, are excellent. In ours the unit is 24 beds, in two wards of 12 beds each, and separated by the duty and service section. Each ward has its own sanitary annexe, and can be subdivided into three blocks of four beds, one of eight and one of four, or simply used as a twelve-bed ward.

3. When the walking convalescent stage is reached, segregation according to type in separate convalescent wards. Separate open-air playing areas or separate periods in playing areas for each type.

Probably this provision for the convalescent period cannot be carried out in its entirety. It is likely that, for a beginning at any rate, we shall confine it to younger children. Adults and adolescents rarely suffer from late complications or cross-infections, and they may be treated in moderately large groups, the precaution only being taken of keeping them apart from children. Then, also, the systematic segregation of child convalescents must be limited at first to the most common types, and if there is a large number of types, some of which are poorly represented, it will not be practicable to arrange for complete isolation of these. The children must be under the supervision of one or more nurses, and, with open-air provision and the teaching of personal hygiene in these small groups, no great harm is likely to occur. While the single-bed ward or cubicle has everything to recommend it for the early stages of the disease, I cannot believe in solitary confinement for walking convalescents.

It is obvious that when a hospital has no facilities for such segregation, no progress can be made in the protection of bed patients, except in the direction of more efficient nursing methods, bed spacing *et cetera*, and none whatever as regards those who are up and about. I do not think the day of the moderately large ward has entirely gone. For reasons of economy and ease of working, some at least may be retained in large hospitals. Older children and adults do quite satisfactorily in them, but it is hoped that they will soon be a thing of the past so far as young children are concerned. I hope at Fairfield gradually to convert all of the twenty-bed standard scarlet fever wards into cubicle and subdivisible wards. Our measles and whooping cough wards are already subdivisible. Unless evidence is forthcoming to make it desirable, I do not propose to apply this plan to diphtheria wards, in which a comparatively small proportion of isolation accommodation should be sufficient. The cost of nursing and other services in subdivisible wards is not so great as in cubicle blocks, though rather greater than in twenty- or twenty-four-bed standard wards.

What can we hope to gain by the adoption of these methods? I am optimistic enough to hope:

1. That the great majority of young children with uncomplicated scarlet fever will make an uninterrupted recovery, and be ready to go home after a stay in hospital of little more than three weeks, as is already the case with adults.

2. That the unhappy state of those in whom infectivity is prolonged, now so often punctuated by a succession of complications and cross-infections, will at the least be a rare instead of a common feature.

If these can be realized, the hospital treatment of scarlet fever will be worth while.

COMMON SKIN DISEASES MET WITH IN GENERAL PRACTICE.[1]

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IN writing this paper I have found it much more difficult than I anticipated to decide on subjects that would be of common interest and to know how much to put in, but most difficult of all, to know what to leave out. However, I have taken the risk of insulting your intelligence by choosing the two commonest skin diseases, which everyone comes across almost daily in his routine work and probably knows everything about—eczema and *acne vulgaris*. I shall also mention scabies, which, although not so commonly met with, is nevertheless very important from the point of view of diagnosis.

Eczema and Dermatitis.

It has been said somewhat cynically that the greatest advance made in dermatology in recent years has been to call eczema dermatitis. Now whether this is the greatest advance I am not quite sure, but it is true that a big step forward has been made in the last few years in sorting out, from the group of diverse conditions conveniently labelled eczema, numbers of diseases which were once wrongly included under this heading. Scabies and impetigo were at one time regarded as forms of eczema; what was called *eczema marginatum* not so long ago is now known to be a fungus infection; and various forms of contact dermatitis until quite recently were thought to be eczema. There are still some, however, who insist that there is a certain type of eruption which is a definite clinical entity, and that this only should be regarded as eczema; but, as we shall see directly, histologically there is no difference between the papulo-vesicle of eczema and that of dermatitis. The most rational outlook to adopt is to limit the use of the term eczema to certain forms of inflammation of the skin in which the cause is not evident, and to regard those with an obvious cause as dermatitis. So I have used the heading eczema and dermatitis, and in the following discussion shall use these terms synonymously.

Goldsmith[1] has defined eczema as an inflammation of the skin in which the epidermis is predominantly involved and which is characterized in the early stage clinically by clusters of tiny vesicles, and histologically by intercellular and intracellular œdema. The most reasonable classification of eczema is based on its histopathology, and so I turn to a brief discussion of this.

The changes which occur are as follows: (i) There is a dilatation of the papillary vessels and œdema in the upper part of the corium (erythematous eczema). (ii) Cellular infiltration around the capillaries follows with increasing œdema, which spreads to the epidermis (papular eczema). (iii) The œdema increases in the prickle cells and between them to such a degree that some of the cells rupture, as also do the intercellular fibrils, and the collection of serous fluid forms microscopic vesicles. These minute vesicles increase in size and are pushed up towards the surface; they coalesce and become sufficiently large to be seen with the unaided eye (vesicular eczema). (iv) The covering horny layer is ruptured either through scratching or from increased tension and the serous fluid escapes (*eczema madidans* or weeping eczema). (v) As the weeping stage subsides the discharge becomes thicker, coagulates, and together with cellular debris forms crusts (crusted eczema). (vi) The vesicles and weeping surfaces may become infected, with the formation of pustules and thick crusts (impetiginized eczema). (vii) As the activity of the process subsides, further cellular division takes place in the prickle cell layer as well as normally in the basal layer, the epidermis becomes thickened, the horny cells are imperfectly cornified, retain their nuclei, remain moist and result in scale formation (squamous or scaly eczema). (viii) From this stage the process may and often does go on to a complete cure, but in some, mainly as a result of constant rubbing, it passes into a chronic state, in which there are a pronounced thickening of the horny and prickle cell layers, a dense cellular infiltration of the corium and greatly dilated papillary vessels. This is the so-called lichenified eczema. These thickened patches are pigmented and often scaly, and the normal furrows and ridges of the skin are exaggerated. It is intensely irritable, and the constant rubbing aggravates the condition.

From the foregoing it will be seen that any particular form of eczema is not a separate entity, but may be regarded as one stage in the evolution of an inflammatory reaction in the skin, the manifestation depending on the nature and duration of the exciting irritant.

Ætiology.

McLeod and Muende[2] have recently clarified the position on the vexed question of the nature of eczema and my comments follow their line of thought. Several theories have been put forward for a number of years and all of them have been discussed from the clinical side without solution: (*a*) that eczema is an entity of local origin and due to local irritation; (*b*) that it is caused by toxins circulating in the blood apart from local irritants; (*c*) that it is simply a form of dermatitis indistinguishable from that caused by local irritants, such as those responsible for occupational dermatitis; (*d*) that there is some constitutional factor that renders the skin sensitive to the causal irritant.

If we approach the problem from the histological point of view, we find that the papulo-vesicle occurring in dermatitis, produced by local irritants, such as turpentine or *Primula obconica*, is indistinguishable from that found in the type of lesion which would ordinarily be diagnozed as eczema. Now, in

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 25, 1937.

urticaria histological changes occur similar to those enumerated above, but in this instance they occur in the corium, and in some cases the reaction is of such intensity that it reaches the epidermis and vesicle formation occurs. Sir Thomas Lewis has shown that urticaria is due to the liberation of a substance of the histamine type, which he calls H-substance, and it is equally possible for this substance to be produced by mechanical injury or through the action of some toxin which has reached the skin by way of the general circulation. It seems quite reasonable to suppose, therefore, that eczema, which is a similar histological process, may be produced not only by external irritation, but by the circulation of a toxin in the blood reaching the epidermis through the cutaneous vessels. In each case the irritant, whether it is external or internal, causes the release from the epidermal cells of some unknown histamine-like substance, which stimulates the cells to proliferate and also provokes a flow of tissue fluid into the intercellular spaces. These two responses are protective in character, the proliferation producing a thicker layer which shields the tissue beneath, and the œdema assisting in the dilution of the irritant. In addition to this exciting factor or irritant it is necessary to have first of all a predisposition or sensitivity of the epidermal cells before an eczematous reaction can result. There is an inherited familial tendency in some individuals to become sensitized, and, according to the situation of the sensitized cells, there may result asthma, hay fever, eczema, urticaria *et cetera*. In eczema the sensitized cells are in the epidermis, but the nature of the process which brings about the sensitivity is at present unknown.

Treatment.

The first essential in the management of all cases of eczema is to try to ascertain whether an attack has been brought on by contact with or ingestion of an irritant, and in this connexion hardly anything is beyond suspicion; it is often something the patient has been exposed to for years and to which he has suddenly become sensitized. If an offending factor can be discovered and its removal effected, unless the condition has been present for a long time it usually subsides quickly and the prognosis is correspondingly improved.

In eczema, as in all skin diseases, any impairment of the general health should be corrected. The bowels must be regulated, dyspepsia remedied, foci of infection eliminated, and a rational, well-ordered routine established.

It is important to remember that the skin is not only an outer covering, but that it is an organ; and especially must it be noted that it is developmentally part of the central nervous system. This is important, because mental disturbances, nervous strain or worry nearly always aggravate, and in some cases appear to originate, many inflammatory skin diseases. Thus physical and mental rest must be aimed at in the treatment of eczema. In the acute and extensive cases rest in bed is essential, for it facilitates the frequent and proper application of remedies, prevents friction from clothes, and enables larger doses of sedatives to be employed to control the itching; and in all cases, both acute and chronic, mental rest must be ensured as far as possible. The main consideration of the patient with eczema is to be relieved of the pruritus; apparently pain is more pleasant to endure than itching, and so scratching, which produces pain, is indulged in to tide over a spasm of irritation. Unfortunately a vicious circle is set up, for the rubbing aggravates the eczema and subsequently the pruritus. It will be seen, then, that sedatives must play an important part in the therapy of eczema; and of the many available I prefer the well-tried bromides, chloral or phenazone.

The diet should be plain and simple, and any food known to aggravate the condition should be eliminated. All condiments and seasoned dishes must be avoided, also very hot drinks and alcohol. A salt-free diet has been advocated, and some experimental evidence has been advanced to show that it is the sodium ion which aggravates the condition. It is certainly worth while to reduce the salt intake greatly or to use one of the substitutes for sodium chloride.

The question of baths and washing is always raised, and in the past soap and water have been strictly banned in all cases. In acute and subacute cases water and especially soap are definitely harmful and often precipitate a recurrence. On the other hand, in the more chronic types, in which the lesions are dry, scaly and thickened, baths (preferably bran, starch or oatmeal) are often found to be very soothing and beneficial, and are much appreciated by patients who have used oil only for cleansing purposes over a long period.

The local treatment of eczema is most important, and the number of applications recommended is legion; but I believe that the general practitioner will obtain better results in all skin diseases if he chooses a few of the common basic prescriptions, learns their action and uses them intelligently, and does not resort to the haphazard use of many. In eczema especially it is not a matter of using an ointment, paste or lotion indiscriminately, for there are definite indications and contraindications for each type of application.

The general principles which should be adopted are as follows: (i) In the erythematous or vesicular stage use powders or lotions. A good powder is the *Pulvis Zinci et Amyli* (A.P.F.) with camphor 1% or 2% added, and for a lotion it is difficult to improve on *Lotio Plumbi* as a wet dressing, or on *Lotio Calaminæ* with or without the addition of *Liquor Plumbi Subacetatis* and *Liquor Picis Carbonis*, which should be dabbed on and allowed to dry. (ii) Weeping stages require lotions, liniments or creams. Lotions as indicated above may be used. *Cremor Calaminæ Liquidus* (A.P.F.) with or without the phenol it contains is very good, as well as *Cremor Zinci*. On all inflamed surfaces it is impor-

tant to have as little covering as possible; certainly no waterproof protection should be used. (iii) Crusted or scaly lesions require starch poultices if the crusts are thick, and ointments if the scales predominate. Ointments, however, must be used with care on an inflamed skin, because they have a greasy base which obstructs radiation and therefore tends to heat the skin and prevent evaporation of water. They also soften the horny layer and sometimes macerate it. A suitable ointment is one containing 2% *Hydrargyri Ammoniatum* and 4% *Liquor Picis Carbonis* in soft paraffin. (iv) Infected lesions require baths, fomentations or starch poultices to remove septic matter, followed by non-irritating antiseptic lotions. (v) The subacute stages, that is, when weeping has subsided, require pastes which have the solid ingredients and a greasy base in about equal proportions. Pastes are less heating than ointments, they do not cause maceration of the epidermis, they adhere well and they form a protective covering; they are also able to absorb a small amount of fluid. Zinc paste is the great standby, and to this may be added 2% salicylic acid if scales are present, and 6% *Liquor Picis Carbonis* to relieve irritation. (vi) In chronic and resistant cases reducing agents are necessary, usually in a zinc paste base; such reagents are prepared coal tar (1% to 6%) or pyrogallic acid (0·2% to 0·4%).

X Ray Therapy.—X rays are of the greatest value in clearing up subacute and chronic eczema. In the subacute type the irritation is allayed, oozing ceases and the thickening frequently disappears in a few days, while lichenified patches of many years' duration will vanish with a few fractional doses of X rays in an extraordinary manner. In fact this is frequently the only satisfactory method of treatment, and patches which have resisted all local applications seldom fail to respond to radiation therapy; it is the most useful single agent we possess in the treatment of eczema that has passed beyond the acute stage.

Prophylaxis.

A patient who has once had an attack of eczema should protect his skin from outside influences more than the average individual. He should avoid hot sunshine and both hot and cold winds. In bathing he should use a super-fatted soap, and for preference he should cover the areas that have been affected with soft paraffin in order to water-proof them. He may use cold cream for cleansing purposes; and in general he should avoid anything which he knows from experience will aggravate or precipitate an attack.

Acne Vulgaris.

It is very doubtful whether *acne vulgaris* is a disease *per se*, and many regard it more as an inborn characteristic, but it is probably part of a syndrome in which there are a number of factors involved. Nevertheless, it occurs with such great frequency in varying degrees of severity, from the presence of a few comedos to the large subcutaneous papulo-pustules of *acne indurata*, and it causes so much distress of mind and mental depression in both young females and males, that it must rank as one of the most important skin conditions with which the general practitioner and the dermatologist alike have to deal. Its frequency may be judged from the investigations which Bloch[3] carried out on 4,191 school children between six and nineteen years of age. The sexes were equal in number, and he found that 64% were affected in some degree.

In view of the fact that there is an unlimited amount of material on which to work, and of the vast amount of research which has been carried out on this condition, it is surprising to find that there is still a considerable divergence of opinion as to its ætiology. Two main views are held as to the cause of acne: (i) that it is an infection of the hypertrophied sebaceous glands; (ii) that the pilosebaceous apparatus is activated by stimuli from the secretion of the gonads at puberty, or that there is some other glandular dysfunction.

Ætiology.

The Bacterial View.—In some individuals there is an inherited tendency to a greasy skin, and in these people the sebaceous glands hypertrophy at puberty, their secretion is increased, and the acne bacillus, which flourishes in a greasy soil, invades the necks of the follicles. Acting mechanically or as a toxin it sets up an irritation, and the epithelium deals with it as it deals with any foreign body, by encysting it with horny cells. The resulting hyperkeratosis at the mouth of the follicle thus blocks it, causing a retention of sebum, and at this stage the obstructed follicle becomes clinically a comedo. A further invasion with staphylococci takes place, which results in an inflammatory reaction occurring around the follicle, with subsequent formation of papules and pustules. This view, however, is gradually losing ground; and at most bacterial infection is looked on as a contributory factor and is considered by many to have no influence at all. Goldsmith,[4] in a systematic investigation, found that no variety of staphylococci was found in acne pustules that was not present in equal or greater numbers on normal skin; nor could he incriminate the acne bacillus, and he says that it has never been possible to produce acne lesions in man by rubbing in cultures of the bacillli.

The Glandular View.—It has for a long time been recognized that a relationship exists between acne and an altered function of the endocrine glands. The onset of acne is associated with puberty; there is frequently a recurrence of the condition at menstruation; eunuchs are said to be immune; sexually over-developed children show a higher percentage of acne than those not so developed, and so on.

Recent experimental work[5] on the estimation of the amount of œstrogenic substances in the blood of patients with acne and of normal females shows that a definite relationship exists between these substances and the development of acne, and the indications are that a deficient secretion of the

follicle-ripening hormone of the ovaries is the direct or indirect cause of acne. From clinical observation and experimental evidence, therefore, it would seem that certain internal factors, for example the physiological and pathological activities of the endocrine system, bring about changes in the follicles and sebaceous glands of the skin; toxic substances resulting from some minor disorders, such as constipation, gastro-intestinal disturbances and foci of infection, play a more or less important rôle as contributory factors; an inflammatory process results, represented in acne by infiltration and suppuration. Infection, if it plays any part at all, does so only in a minor or secondary capacity.

Diagnosis.

Diagnosis is usually easy, because some blackheads are nearly always present; but in some protracted cases the condition may merge into and become associated with rosacea, and it may not always be easy to make a decision between the two. Acne practically never goes beyond the age of thirty years and blackheads are always found, whereas rosacea is seldom seen earlier than this age and comedos are never seen. Acne affects the sides of the cheeks, the temples and the chin, while in rosacea there is intense flushing of the face with papulopustules on the nose, the centre of the forehead and the chin.

Treatment.

Acne vulgaris is essentially a syndrome of puberty, adolescence and early adult life. It is to a certain extent a self-limited disease, and it tends to disappear of its own accord. In the milder cases the lesions have gone by the time puberty is fully established, and in these very little scarring results; but in many the condition drags on even up to the age of twenty-five years, and these unfortunate individuals not only have to contend with the associated disfigurement during so many years, but are usually left with a permanently scarred skin. I therefore contend that even though the condition may be physiological and of comparatively short duration in some, the mildest cases should be treated with the appropriate measures in order to try to limit the amount of ultimate scarring. Measures directed towards improvement of the general health should not be overlooked, and correction of the so-called minor disorders, such as constipation, gastro-intestinal disorders, anæmia and foci of infection, is important.

Diet.—Just what the cause and effect is of certain articles of diet on acne seems very speculative, but some foods, for example chocolate, cocoa, cheese, pig flesh, pastry and excess of carbohydrates, are undoubtedly important factors. Chocolates in particular are especially harmful, and it is a common experience to hear young girls blame a chocolate debauch for a recurrence of their acne; some even go so far as to say: "One chocolate, one spot."

Internal Remedies.—Numerous drugs have been recommended from time to time in the treatment of acne, but none has withstood the test of time. However, calcium sulphide is a popular remedy with some, and in a few cases does seem to have a beneficial effect; it should be given in doses of 0·06 to 0·12 gramme (one or two grains) three times a day, one hour after food.

Considering the modern view of the pathology of acne, glandular therapy presents theoretically an enticing field, but practically has so far been disappointing in its results. Much work has been done on this problem, and although some authors claim good results with pituitary extract and "Antuitrin S", others again have failed to substantiate their findings. In spite of the disappointing results, this would seem to be the field in which progress in treatment is likely to be made; and when more is known of pituitary and gonadal dysfunction, it is probable that a more satisfactory means of therapy will be at our disposal. Vaccines are of little value, and staphylococcal toxoid has proved ineffective in the majority of acne cases in which it has been used. This is not surprising, and it goes to confirm the belief that bacterial infection is not the cause of the disease.

External Applications.—In our attack on acne we still have to rely mainly on the use of external applications, and the most important of these are soap and water. Regular washing of the greasy skin with soap and hot water at least twice a day is essential; medicated soaps are often advised, but, apart from the psychological effect and the fact that a special soap will be used more frequently than a plain one, they can hardly have any specific effect. After the skin has been washed, a lotion consisting of 1·2 grammes (20 grains) each of zinc sulphate and sulphurated potash should be applied, and when dry the powder may be lightly massaged into the skin. If a paste is preferred, a satisfactory application may be made of resorcin and precipitated sulphur, 0·6 to 1·8 grammes (10 to 30 grains) of each, in one ounce of zinc paste, according to the drying effect desired. If the skin desquamates too freely, or if an inflammatory reaction is set up, the lotion or paste should be omitted for a day or two and then used again with less frequency. The object is to bring about a mild degree of exfoliation, which opens up the follicles and prevents accumulation of secretion.

Comedos, especially if large, should be removed, preferably with an expressor; squeezing them out with the nails often brings about a secondary infection and increased scarring. The large indurated pustules are best treated by puncturing the centre with the point of a small tenotomy knife and evacuating the contents; they then speedily resolve. The associated greasy seborrhœic scalp must not be overlooked; it should be shampooed at least once a week, and a lotion containing 0·6 to 1·2 grammes (10 to 20 grains) of salicylic acid and 0·03 gramme (half a grain) of perchloride of mercury to the ounce of industrial spirits should be applied once a day.

X Ray Therapy.—Many cases respond very satisfactorily to persistent and regular treatment with local applications and with attention to the general health and diet, but even then there still remain a comparatively large number who, after the most conscientious endeavours along these lines, fail to show the desired improvement. To these patients X ray therapy has proved to be of inestimable value and can safely be said to have revolutionized the treatment of acne. All cases, and especially those occurring during adolescence, should first of all be tried with local and general measures; but if improvement does not occur, then X ray therapy should be resorted to without delay, for this treatment is undoubtedly, when properly administered, the method which to date gives the most satisfactory results in the resistant cases.

I shall not discuss details of dosage and technique. The machine used should be calibrated, the dosage should be accurately measured and the intervals of exposure should be estimated according to the type and severity of the case. It is therefore perhaps hardly necessary to mention that irradiation treatment should be undertaken only by those who are thoroughly acquainted with the therapeutic use of X rays, and who have sufficient experience to know when and how it should be administered.

To sum up, each case of acne should be treated on its merits. Vigorous measures, especially with local applications, should be undertaken in its early stages, and X ray therapy should be used when these measures have failed to arrest progress of the complaint.

Scabies.

I have chosen scabies for discussion because it is not altogether rare; and, owing to the frequency with which it is associated with complications, the diagnosis is often very difficult and cases are frequently missed. It has been said that the diagnosis of scabies is at once the easiest and the most difficult in dermatology. Stokes[6] has emphasized the difficulty in diagnosis in a recent review of 53 cases from among the well-to-do. He found that 37 patients had seen one or more doctors without relief; eight had seen "Grade A" dermatologists with five errors in diagnosis; and only ten correct diagnoses had been made by 49 physicians.

Many errors are apparently made because it is not generally recognized that scabies is wholly without social boundaries and that cleanly people and those placed high in the social scale are by no means immune from attack. The disease may be contracted in any circumstances when contact with an infected person has been established, but nevertheless in a patient of hygienic habits one hesitates to make the diagnosis of scabies unless the acarus can be demonstrated, and this is by no means always easy.

Diagnosis.

In considering the diagnosis of scabies it must be remembered that the original and typical lesions may be completely or almost completely obscured by complications, such as pyogenic infection, a secondary dermatitis due to scratching or to too vigorous medication, and urticaria. Post-scabetic urticaria is an interesting complication and is probably due to a sensitization to the proteins of decomposing acari, their excreta or toxins. It appears four to six weeks after infection, and may remain for months after the scabies has been cured.

There are four main characteristics that should be taken into account in the diagnosis of scabies: (i) The presence of burrows and the demonstration of the acarus naturally clinch the diagnosis. (ii) Nocturnal itching is very suggestive, but most irritable dermatoses are worse at night. (iii) The source of contact can frequently be found, and others in the house are usually affected. (iv) The distribution of the eruption is significant. Pels[7] emphasizes the importance of distribution of the lesions in the absence of definite burrows or demonstration of the parasite, and says that scabies definitely lends itself to diagnosis from a topographical standpoint. It is necessary for the patient to strip completely, and the diagnosis can then often be made or strongly suspected from a distance. In the male the affected areas are the genitalia, the palms, the fingers, the flexor aspects of the wrists, the axillary folds and the buttocks. In the female the areas are the same, except that the genitals are free, but the breast areas, especially the upper quadrants, are involved. The two most important areas are the male genitalia and the upper portions of the female breasts. It should be noted in passing that the distribution in infants is atypical; every part of the body, including the face and scalp, may be involved. The soles of the feet are commonly affected, and also the back of the neck, which rests on the mother's infected wrist. To sum up, any irritable eruption between the levels of the breasts and knees should be suspected as scabies, and even in the absence of burrows or acari a therapeutic test is justifiable.

Treatment.

Intensive treatment with sulphur ointment over two or three days is a widely used and very satisfactory therapeutic measure; but a new method[8] has recently been advocated, which brings about the precipitation of colloidal sulphur onto the skin by the interaction of sodium thiosulphate and an acid. After the patient has had a good bath and has been thoroughly dried, a 40% aqueous solution of sodium thiosulphate is applied over the entire body except the head and face. Fifteen minutes later a 4% solution of hydrochloric acid is applied similarly, and one hour later the applications are repeated in the same order. The same procedure is carried out next day, and on the third day a bath is taken, clothing is changed and all linen *et cetera* is sterilized.

The advantages claimed for this method are that it is simple and easy to apply, that the risk of sulphur dermatitis is reduced to a minimum, that there is no objectionable, prolonged treatment with greasy applications, and the percentage of cures is greater than with other methods.

I have tried it only in three cases. One patient was cured with one treatment, another disliked the dryness of the skin produced and did not respond satisfactorily, and the third did not report again, so I hope was cured. It seems an attractive form of treatment and, I think, is worthy of a further trial.

Conclusion.

I have obviously not attempted a complete review of the above disorders, but have dealt with a few aspects which I thought might be of interest to those doing general work. Skin diseases can no longer be regarded as a thing apart, and it is essential for close cooperation between physician and dermatologist in order that the best interests of the patients may be served.

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THE COMING OF MEDICINE TO TASMANIA: SOME EARLY MEDICAL MEN AND THEIR ENVIRONMENT.[1]

By W. E. L. H. CROWTHER,
Hobart.

"Who shall go over the sea for us and bring it unto us, that we may hear it and do it?"—Deuteronomy XXX, 13.

BY the commencement of the nineteenth century the settlement of Port Jackson was well established and ships from Europe and America were frequent visitors to the Australian coast. They came to explore or to take the whales and fur-bearing seals. Governor P. G. King, fearing an invasion of his authority, was considerably embarrassed by these arrivals. By reason of its long-continued and detailed survey of the coasts of southern Australia, the expedition of Baudin, following so soon that of D'Entrecasteaux, particularly aroused his fear. To forestall annexation of any part of his dominion, he dispatched Lieutenant Bowen, R.N., to form a settlement at the Derwent. A small coasting vessel was also sent to King's Island, where, in the actual presence of the French, possession was proclaimed. Bowen made his landing at Risdon Cove, on the eastern side of the Derwent River, on September 7, 1803, and was followed by the larger expedition of Lieutenant-Colonel David Collins, which anchored off his camp during February, 1804. Considering Risdon to be unsuitable for a permanent settlement, Collins, on February 20, formed his camp at Sullivan's Cove, the present site of Hobart.

Following the withdrawal of Bowen's party a landing was made on the northern side of the island, where, on November 11, 1804, Colonel W. Paterson hoisted the colours and, with the usual ceremony, inaugurated his *régime*. With Paterson at Georgetown as surgeon was Mr. Jacob Mountgarrett, R.N., late of His Majesty's ship *Glatton*. He must be considered as the pioneer medical man of this State, having landed with Bowen as surgeon at Risdon. Thus, by the end of 1804, settlements were established at Hobart Town and Georgetown. These were, however, quite independent of each other; their respective commandants were responsible only to their superior at Sydney.

In an earlier address[1] some medical aspects of the settlement of the Derwent were considered, particularly the medical personnel concerned, an outbreak of scurvy and the equipment of the hospital. Until December, 1807, both settlements were on a very reduced ration, and at some periods were on the verge of famine. To meet the situation convicts and others were allowed to go to the bush as whole-time hunters of the kangaroo and emu. With the meat thus brought in and with scanty supplies from Port Jackson and India, the little communities struggled through until able to support themselves.

With Lieutenant-Colonel Collins had landed a guard of the Royal Marines, some free settlers and medical and civil officers to administer the convict establishments. His medical staff were William I'Anson, surgeon, Matthew Bowden, first assistant, and William Hopley, second assistant surgeon. The two first-named were each in their twenty-fifth year and Hopley was probably no older. They were paid £182 10s., £136 17s. 6d., and £91 5s. respectively. The Lieutenant-Governor, in a dispatch, makes this allusion to them:

> I feel myself very much at ease with the medical aid that has been provided for me and do not consider that for some time to come any addition to them will be required.

Collins himself, an able soldier and administrator, was embittered by the long neglect of the Colonial Office; yet he was approachable and considerate to his officials and shared with them what social life was to be had.

The Reverend Robert Knopwood in his journals notes considerable visiting and dining among the little staff, especially when the arrival of one of His Majesty's ships or a south sea whaler meant news from home, new persons to meet, and some addition to the scanty supply of rum and spirits in the camp. The first break in the medical establishment was when, on September 10, 1808, Mr. Hopley was reported upon as medically unfit, and was invalided to England. His experience had not been a happy one. The only married man of the three,

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

he had been "near three years under damaged canvas, and at last obliged to build a home for his family at a very great expense, no barracks being even yet erected for officers nor any remuneration in lieu" (statement, March 12, 1810). Suspended by Collins for misconduct in 1807 and later, after a severe reprimand, reinstated, he left the colony in the following year, only to return in 1810. In a memorial to Colonel Macquarie resigning as assistant surgeon and applying for a grant of land, he mentions his services and the fact that he had had the care and upkeep of his mother-in-law (the widow of Lieutenant Hobbs) and her six children in addition to his own family. His four sisters-in-law, however, he states were now married to officers

Figure I.
Hobart Town in 1821 (Evans).

of the Derwent settlement. After Hopley's death some years later there was so little provision for his widow that his two sons were in 1820 approved for admittance to the male orphan institution at Hobart Town.

On March 24, 1810, occurred the unexpected death of Lieutenant-Governor Collins at the age of 56. He died suddenly while sitting in his chair and talking to his attendant. Except for a slight cold, he had had little warning of his end, which was hastened by worry connected with insubordination of his marines and the protracted stay of Captain Bligh in the Derwent.

William I'Anson had died before February, 1812, as on that date Mr. Bowden was promoted to surgeon *vice* W. I'Anson, deceased. Of the latter there is little to record beyond odd reports of his in the historical records of Australia which tell of minor activities at the settlement and detail two lists of the sick in the hospital with their complaints.

Matthew Bowden survived his chief less than three years. He, too, had had variations of fortune. Apart from his duties as assistant surgeon, he had farmed 100 acres of land on which, in 1806, he had one cow and bullock and twenty-one rams and sixty-eight ewes, as well as two male and five female goats. In a private memorandum to Lieutenant-Governor Davey, dated February, 1813, Colonel Macquarie cautions him to be on his guard against some designing characters at the Derwent, who will endeavour to impose upon him and mislead his judgement by artful insinuation and plausible but interested projects and speculations. Bowden, I regret to say, is among the persons mentioned. Knopwood gives the following considerable information as to the manner of his death and burial.

October, 1814.

Friday, 21st. I dined at home late. Took some wine with Mr. Birch. The Governor there. Mr. Smith smoked a pipe with me. Mr. Bowden went to his farm to shear some sheep.

Sunday, 23rd. Performed divine service, attended by His Honour the Lt.-Gov. and family and a large congregation at the new barracks. Just as I had finished the prayers we received information that Mr. Bowden was

dead and that he had died on his way home from his farm to Hobart Town. So sudden a death causes uneasiness to all who knew him, but especially to those who came out with him when the late Lt.-Gov. Collins came out. The only surviving officers are Mr. Hopley the surgeon and myself. God only knows how long I may continue. I have had bad health for some time. At three o'clock the body was brought over in a shell. All the officers met him at the landing and walked up to the house with it. . . .

Friday, 28th. Busy in preparing for the funeral of poor Mr. Bowden, the principal surgeon of the colony.

Saturday, 29th. The day very fine. Busy preparing for the interment of Matthew Bowden, Esq. (A plan here follows of the order of the procession, with the officials, military and inhabitants generally.)

Sunday, 30th. The body was interred at half-past five p.m. We returned to the house for a very little while. . . . Performed divine service, attended by all the civil and military officers. I preached a very excellent sermon on the "death of our parents" (Zach. V, 5). Your fathers, where are they? I received thanks of Mrs. Davey, Capt. Clark and many other officers. . . .

The tombstone is in David's Park and still in excellent preservation, and has this inscription:

Sacred to the memory of Matthew Bowden, Esq., late principle servant of the colony, to which he came with the late Gov. Collins. Departed this life October 25th, 1812, aged 35 years and 12 days, leaving a family of two children with a disconsolate mother to lament the loss of their dear protector, who fulfilled the duties of an affectionate husband and a tender father and a faithful friend.

In the early part of his life he had had the honour to bear His Majesty's commission as surgeon to the Royal Lancashire or King's Own Regiment, under the command of Sir H. Haughton, and after faithfully and honourably conducting himself, so as to gain the admiration of his country, the awful hand of death summoned him to an immortal crown of glory.

> All earthly honours soon or late must die
> And on the silent tomb neglected lie,
> That Christian work and virtue so renowned
> In glorious immortality is found.

In Knopwood's description of Mr. Bowden's funeral among the "Officials and gentlemen of the Colony" immediately following the coffin was a Mr. Birch. Mr. Thomas W. Birch had been formerly surgeon to the English south sea whaler *Dubuc*, and subsequently became medical man and merchant adventurer of Hobart Town; in fact our first medical "truant". His ship, the *Dubuc*, had put into the Derwent in 1808 in an unseaworthy condition, and having been condemned, was beached at Kangaroo Point, where for many years the rotting timbers of the old whaler were to be seen. Mr. Birch is said to have brought out with him a cargo of goods and with the profits of their sale to have established himself as a merchant in the town. In a medical capacity we have only one record of his activity, when he was directed by Collins, with I'Anson and Bowden, to inquire into the health of William Hopley, second assistant surgeon. Their report, dated September 10, 1808, records that they have "reason to believe a removal to his native climate might be of benefit to his recovery, and we do therefore beg leave to recommend his being allowed to return to England for that purpose."

There is a family tradition that Birch gave advice gratis to the poor of the town. When, in 1820, Mr. Commissioner Bigge took evidence (in a ward of His Majesty's colonial hospital by the way) on the state of trade and conditions generally in Van Diemen's Land, Mr. Birch was one of his principal informants. There is a *verbatim* report of his evidence in the Historical Records of Australia. It is stated there that he had carried on whaling almost every year from 1808 (at this period the southern Right whale visited the bays and estuaries of the island in order to calve, and the whaling season extended from May to November of each year). He was the owner of at least three ships, the *Henrietta Packet*, the *Lachlan Macquarie* and *Sophia*. The first, a schooner of 60 tons, was built in 1812 by Samuel Gunn "on the stocks" at the Derwent. The *Lachlan Macquarie*, a square sailed vessel, was launched in the same place in the following year. His brig the *Sophia* was purchased by him in 1816 and remained in his possession until 1821, when after his death it was bought by the Government and renamed the *Duke of York*. Afterwards it was used as a powder hulk, and then it was moored in Sullivan's Cove as a home for the chain gang.

Birch's shipmasters were Captains James Kelly and Feen, both brave and able men who played a considerable part in the development of the colony. In December, 1815, Birch fitted up an open whale boat, the *Elizabeth*, in which Kelly, with a crew of five men, circumnavigated Tasmania. A full account of this voyage is given in Kelly's journal, with details of the discovery of Port Davey and Macquarie Harbour. Feen, in 1816, with the *Sophia*, was actively engaged in whaling on the Derwent, and obtained sixteen tuns of oil, whilst Kelly, with the *Henrietta Packet*, was away on a sealing voyage. Fired by Kelly's discoveries, Birch later in the same year accompanied him in the *Henrietta Packet* when Port Davey was explored. On their return the little schooner was sold to Denis McCarthy. The *Sophia*, under Kelly in 1817, made three voyages in three months to Macquarie Harbour for Huon pine, Birch having been granted a monopoly of the taking of this valuable timber for one year as a reward for his discoveries. At the end of the year Kelly took the brig sealing to New Zealand, and their adventures there, culminating in bloody fighting with the "Moureys" (as Kelly called them), are an interesting part of the history of New Zealand. Full details of this affray were given on his return in the *Hobart Town Gazette* (March 28, 1818).

Back in Hobart with 5,000 seal skins, the *Sophia* was most fortunate in the 1818 whaling season, taking in three weeks 35 tuns of oil and on the second venture six more whales. On one of several trips for Huon pine in 1818-1819, Mr. Florence was a passenger and carried out a survey of Macquarie Harbour, and subsequently reported on its resources to the Government. In this year a band of convicts captured the Government launch on the Derwent and escaped to sea. Mr. Birch at once offered the brig for the pursuit and followed with a party of the Forty-sixth Regiment. Although they avoided

the *Sophia*, the convicts were later recaptured among the Bass Straits islands. For this service Birch received the thanks of the Government and a remission of the duties on a quantity of spirits imported by him. Previously there had been a conspiracy among a party of bushrangers to make their way to Macquarie Harbour, capture the *Sophia* and make their escape overseas.

Other activities of the brig were to convey parties and goods to Port Dalrymple and Sydney. Birch himself is mentioned in the Historical Records of Australia as having cured disease in Mr. Loane's sheep by the use of tobacco (the latter he supplied himself at 20s. per pound, a very high price for that time). His mansion, Macquarie House (for many years the Macquarie Hotel), was built in 1815. The first house in the colony to be built of brick, it was of three stories and had an embrasured parapet. For a time used by Lieutenant-Governor Sorell as Government House, and later as headquarters of the military during the Black war, it has served over a century as hotel, boarding house or private residence and trades hall. It is now, with a new front, in use as flats and medical rooms. Adjacent to Macquarie House Birch owned a large block of land, and Saint Joseph's Church stands on what was his vegetable garden. As well as building seven other brick houses, he was the contractor for the brick wall around the old gaol. His death occurred at Macquarie House on December 1, 1821, and the family vault was desecrated after his burial.

Mrs. Birch married again and had to endure a spendthrift husband dissipating her fortune (for she had been left an estate that realized at auction in 1838 the sum of £40,000). The Court, to protect her interests, ordered a sale of the estate when, in addition to the property already mentioned, a tannery with 100 acres of land in Upper Macquarie Street, as well as eight farms, which included Lovely Banks at Jericho and the Duck Holes at Richmond, were disposed of. Other assets had been his timber mills and property at Birch's Bay (D'Entrecasteaux Channel) and the *Sophia*.

His name is still recalled by Birch's Avenue, off Macquarie Street, Birch's Inlet at Macquarie Harbour, and the bay just mentioned. By his enterprise in exploration and commerce he ranks with Campbell, of Sydney, as one of the two principal merchant adventurers of the early days of Australia.

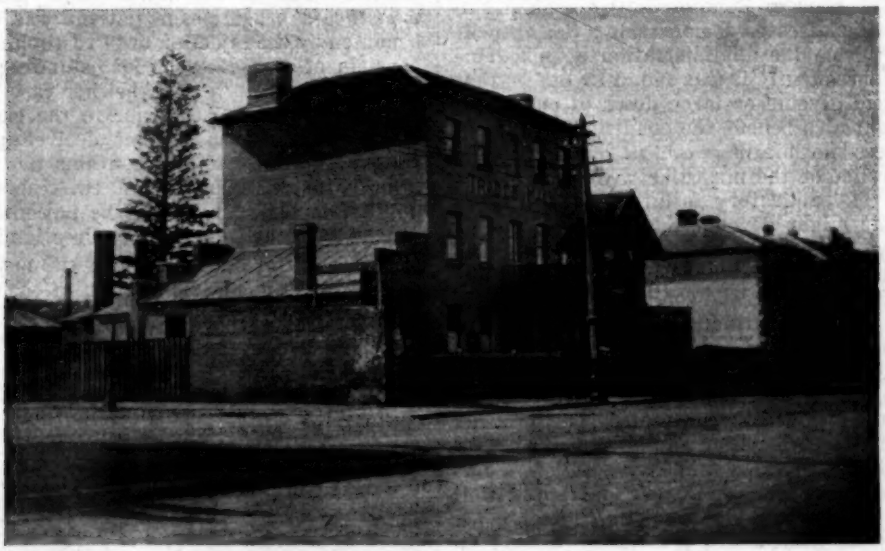

FIGURE II.
Macquarie House, Hobart.

Acknowledgements.

Two principal sources of information have been drawn upon—the "Historical Records of Australia" (H.R.A.), Series 3, Volumes I to IV, and the journals of the Rev. R. Knopwood. The latter are incomplete; there is an important gap from 1808 to 1814. Those that remain are in private possession or at the Mitchell Library. Full details of the activities of Captain James Kelly on the coasts of Van Diemen's Land and New Zealand are given in a series of articles he contributed to the *Hobart Town Courier* in 1855. Descendants of Mr. Bowden and Mr. Birch have also given me interesting details as to their ancestors.

For the interesting photograph of Macquarie House I have to thank R. H. Robinson, Esq.

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THE RELATION OF MYASTHENIA GRAVIS AND ALLIED CONDITIONS TO "PROSTIGMIN" THERAPY.

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MYASTHENIA GRAVIS is an uncommon condition. Until recently its recognition in general practice may not have seemed of great importance, for no measure other than rest was of value in its treatment.

In 1930 Dr. Harriet Edgeworth,[1] herself a sufferer from this disease, demonstrated that benefit might result from the administration of ephedrine sulphate. Subsequently it was found by others that the amino-acid glycine or large doses of potassium chloride might also benefit the condition.

Nevertheless no means were at hand for the immediate relief of the muscular weakness, nor of crises, such as respiratory failure or inability to swallow, that might develop.

Dr. Mary Walker[2] considered the muscular weakness present in *myasthenia gravis* to resemble that occurring in curare poisoning. As physostigmine is a partial antidote for this drug, she employed eserine hypodermically in *myasthenia gravis*. The results were immediate and dramatic. Subsequently the less toxic analogue, "Prostigmin", was substituted,[3] with even greater benefit. This beneficial effect has now been amply confirmed. Not only may the muscular weakness be relieved, but the serious crises of respiratory weakness and failure in swallowing yield to its injection as dramatically as may acute pulmonary œdema to the injection of morphine.

The physiological basis for the employment of eserine is as follows. There is evidence that acetylcholine may mediate the transmission of nerve impulses to voluntary muscle across the neuromuscular junction, and that a specific choline esterase exists in normal blood which may destroy acetylcholine. Should an excessive esterase activity be the cause of *myasthenia gravis*, eserine, which in general inhibits esterase activity, would be expected to relieve the muscular weakness. Investigations, published in a separate paper by Dr. A. B. Corkill and Mr. A. H. Ennor,[7] demonstrate that the blood esterase activity is not increased in *myasthenia gravis*, and therefore the possibility of an increased activity in a local tissue esterase has to be considered. This latter suggestion would better explain the local paralyses, such as those of the eye muscles, which so frequently occur.

Whether all cases of *myasthenia gravis* yield to "Prostigmin" therapy has yet to be proven. Group III, to be described, consists of cases resembling myasthenia in certain respects; but the patients in this group obtained no benefit from "Prostigmin". It does, however, seem likely that the injection of "Prostigmin" offers a ready measure for the diagnosis of this condition in doubtful cases.

The pathological examinations of the central nervous system, or of the muscles of patients who have died with this condition have thrown little light upon its essential nature. Nor does a neurological examination reveal those signs which can be unequivocally associated with organic disease of either the central nervous system or the peripheral nerves. It would seem as if the solution of the nature of the disorder must come from the laboratories of the physiologist and biochemist rather than from that of the pathologist.

The cases to be reported are divided into three groups. The first contains three cases of severe *myasthenia gravis*; each patient was dramatically benefited by the injection of "Prostigmin". Group II consists of two cases of long-standing myasthenia of a less severe type. In neither case had the condition been recognized prior to the employment of "Prostigmin"; each patient derived great benefit from its use. Group III contains two cases resembling mild myasthenia and two cases of nuclear ophthalmoplegia, in which the patients derived no benefit from the injection of "Prostigmin".

The essential details only of the medical history and of the clinical examination of each patient are here described.

Group I: Severe Myasthenia Gravis.

CASE I.—A male, aged fifty-six years, was admitted to the Alfred Hospital on March 12, 1936, with severe *myasthenia gravis* (Figure I). His past and family history were not of importance, except that he stated that he had attended the Eye and Ear Hospital twenty years

FIGURE I.
Case I: Bilateral ptosis and ophthalmoplegia resulting from *myasthenia gravis*.

FIGURE II.
Case I: Restoration of ocular movements and relief of ptosis following the administration of "Prostigmin".

previously, suffering from double vision and slight ptosis. These symptoms then rapidly disappeared. He had remained well and performed manual labour until three months previous to admission. He then became unduly tired at the end of each day's work, suffered from double vision, and his right upper eyelid drooped slightly. Five weeks later he noticed a weakness in his calves; this

rapidly increased until he could walk only with the greatest difficulty. He had noted weakness in the movements of the arms only four weeks previous to admission.

He was found to be suffering from gross muscular weakness, the legs being relatively less affected than the arms. The right eye could be slightly adducted only; the left could be moved upwards and inwards only and could be abducted. A pronounced bilateral ptosis was present.

He was kept at rest and given 0·04 gramme (two-thirds of a grain) of ephedrine sulphate twice a day, with glycine, for five weeks. At the end of this period he seemed worse and was able to swallow fluids only with difficulty. On April 25, 1936, his hand grip, as measured on a dynamometer, was nil; he could just abduct his right arm against gravity to a right angle, the forearm being fully flexed. He could raise his left arm only about 40°. Ptosis was gross and the eye movements were inconspicuous. He could just walk with assistance.

He was given 1·25 milligrammes of "Prostigmin" with 0·6 milligramme of atropine hypodermically. Within ten minutes his ptosis had lessened, his facial expression had altered and his grip was measurable. In half an hour his ptosis had entirely disappeared, his eye movements were full, his arms could be powerfully abducted, his grip was approximately half that of a normal man, as measured by a dynamometer, and he could walk well. His mental state was now one of well-being (Figure II).

This beneficial effect persisted for four hours; it then lessened, and at the end of eight hours all effects had passed. He has since been kept in a fairly comfortable state of health by means of small daily doses of "Prostigmin", although he leaves his bed only after he has had an injection. Eserine, in a dose of 2·4 milligrammes (one twenty-fifth of a grain), was given hypodermically on one occasion. It produced only slight symptomatic alleviation of his condition, with violent vomiting, purging and sweating.

CASE II.—A man, aged forty-nine years, was admitted to the Alfred Hospital on December 9, 1936. He was grossly myasthenic and was suffering from severe thirst on account of his inability to swallow (Figure III). He had seen an oculist seventeen months previously for a drooping of the right upper eyelid. Glaucoma was discovered and he was subjected to operation seven months later. On returning to his work he suffered from general weakness. Three months before admission to hospital he had found that his head would drop forwards upon his chest and he could raise it only by using his hands. A little later his lower jaw dropped, and although he could purse up his lips, his masticatory muscles were too weak to raise his jaw against gravity. About one month before admission his vision had become blurred, and a little later he experienced great difficulty in swallowing and had choking attacks. At about the same time he noticed weakness of the arms and legs. He also found it difficult to breathe. His double vision was only recent and transitory. He had been unable to eat any substantial amount of solid food for some weeks.

FIGURE III.

Gross bilateral ptosis and sagging of the jaw resulting from *myasthenia gravis*.

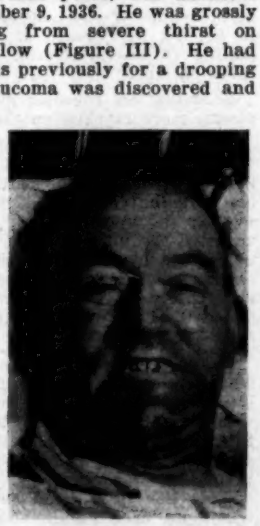

FIGURE IV.

Relief of ptosis and restoration of jaw movements following the administration of "Prostigmin".

He was found to be suffering from severe myasthenia with laboured breathing. Examination revealed bilateral ptosis, impaired movements of the eyes, weak movements of the soft palate, an inability to raise his sagging lower jaw or to raise his head from the pillow. His leg movements were fairly powerful. The arm movements, too, were moderately good, except that extension of the right forearm was very weak. He had several alarming choking fits while in hospital.

He was given 1·25 milligrammes of "Prostigmin" hypodermically. Within five minutes he remarked that his respiratory distress had ceased. At the end of half an hour all his eye movements were full, his ptosis had disappeared, he could raise his head from the pillow and press powerfully forward against resistance and could close his jaw powerfully (Figure IV). This effect invariably followed the injection of "Prostigmin". He was discharged, taking small daily doses of this drug, and is thus able to lead a tolerably comfortable vegetative existence.

CASE III.[1]—A married woman, aged twenty-five years, was admitted to the Alfred Hospital on May 21, 1937, suffering from lead poisoning. She had been well until three years before, when she had suffered a severe scald of the left leg and had spent three weeks in hospital. Since then she had become progressively weaker and at times had seen double. She had been able to do a little work until after the birth of her second child one year before. Since then she had spent much of her time in bed, and often had been hardly able to raise her head. In the light of subsequent examination every effort was made to establish a history of contact with lead. None could be given, although it was obvious that large quantities had entered her system. She was pale and had respiratory distress. Her voice was so weak as to be at times hardly audible. There were a well-marked blue line of the gums, secondary anæmia (the red blood cells numbered 3,340,000 per cubic millimetre and the hæmoglobin value was 52%) and considerable basophilic stippling.

Despite these findings she showed no evidence of peripheral neuritis or that her nervous system was seriously affected by the lead. She was regarded as suffering from severe *myasthenia gravis*. She had bilateral ptosis and a mild ophthalmoplegia in that full vertical and right lateral movements of the eyes were impossible. The palate moved only slightly and the tongue was poorly protruded. There was a myasthenic facial expression, and the angles of the mouth could be retracted only weakly. The movements of the arms were grossly affected, her grip being hardly measurable with the dynamometer. She had been unable even to swallow fluids on the day of examination and was extremely thirsty. Her voice was hardly audible.

She was given 1·25 milligrammes of "Prostigmin" hypodermically. Within five minutes her facial expression had altered, her ptosis had disappeared and her eye movements were full. Her grip was now one-third of that of an average normal woman. Her voice was strong and she was able to swallow freely. In half an hour she was able to walk and move as a normal woman. Her grip was now moderately strong.

She became agitated that night and was given paraldehyde. She was found dead in the morning, having apparently died in her sleep from respiratory exhaustion. The diagnosis of lead poisoning was confirmed at autopsy.

It is obvious that in the severe case of *myasthenia gravis* "Prostigmin" may be life-saving. It is possible that in the third case, had the respiratory

[1] I am indebted to Dr. W. S. Newton for permission to publish an account of this patient, whom I saw on his behalf.

failure, which is presumed to have been the cause of death, been observed, a timely injection might have quickly allayed it.

The two surviving patients are unable to work. Although there has been a recent reduction in the price of "Prostigmin" in Australia, it is still beyond the means of most public hospital patients in quantities sufficient to enable them to remain almost constantly under its influence. In the quantities in which we have injected it, the results have usually passed off after about four hours. However, some beneficial effect has at times remained for eight hours. A dose of from 0·3 to 0·5 cubic centimetre has often proved a useful one. On special occasions it has been repeated during the day. With a further reduction in its price it will be able to be used much more freely. Unfortunately, much larger quantities are necessary when the drug is taken by mouth. It is said that as much as 30 milligrammes are needed to produce the equivalent effects of the injection.[4] Although 10 to 15 milligrammes are said to be useful,[5] the expense in Australia up till now has been prohibitive for the poorer patient. Recently, however, the tablets have been made available at a considerably reduced rate. It is likely, then, that in the future even the patients severely affected may be able to engage in a measure of work.

Group II: Myasthenia Gravis of Long Standing.

In the second group are included two patients suffering from myasthenia of long standing and of a milder type. Such cases should be object lessons to those who have not as yet familiarized themselves with the manifestations of this peculiar disease. It will be seen that, unless inquiry is made, a wrong

FIGURE V.
Case IV: Bilateral ptosis resulting from mild *myasthenia gravis*.

FIGURE VI.
Case IV: Relief of ptosis following the administration of "Prostigmin".

diagnosis may be readily arrived at; it is not unlikely that such a patient may either be lightly dismissed as "neurotic" or subjected to prolonged psychiatric treatment. As will be observed, the use of "Prostigmin" may readily dissociate the true myasthenic from the neurasthenic group.

CASE IV.—A married woman, aged forty-six years, was referred by Dr. M. D. Silberberg as possibly suffering from *myasthenia gravis*. Over twenty-five years ago the late Dr. O. Sullivan was said to have remarked that he could always tell when she was tired by the drooping of her lids. Her mother had noted the same association for at least twenty years. She complained of palpitation and "nerviness", and at times her arms would become so weak that she could hardly raise them. Her legs, too, would give way and she could hardly drag them along. She also

FIGURE VII.
Case V: Bilateral ptosis resulting from mild *myasthenia gravis*.

FIGURE VIII.
Case V: Relief of ptosis following the administration of "Prostigmin".

suffered from pains at the back of the knees and numbness of the hands if she held anything for long, from difficulty in getting her breath on exertion, and from a frequent feeling of breathlessness even while resting. She had three children.

Examination revealed nothing abnormal of note save ptosis, more marked on the left side (Figure V).

Following an injection of 1·25 milligrammes of "Prostigmin" she noted in ten minutes that her eyelids were rising. She then said that her breathing had become normal and she felt that she could now breathe with the lower as well as the upper part of the chest. Her eyelids rapidly assumed the normal position (Figure VI). At the end of an hour she remarked that she felt perfectly well, which she had not felt for years. She has since continued to take this drug with great benefit. She is unable to take ephedrine owing to discomfort produced in her chest. She also takes glycine.

CASE V.—A single woman, aged thirty-six years, was referred by Dr. M. Ashkenasy as possibly suffering from *myasthenia gravis*. Thirteen years before she had fallen when running and again when coming downstairs. After that she had frequently fallen in the street for no apparent reason. Shortly after she noted that her arms were becoming weak so that at times she could hardly raise a cup to her lips. She had undergone a long course of treatment at a public hospital. She had been forced to give up her work seven years before, and had had a long course of massage. Four years before she had undergone psychiatric treatment, including a course of hypnosis. She had then been told that nothing further could be done for her and she had had no medical attention since. As far as could be determined, the true nature of her condition had not been recognized, and she was regarded as suffering from a functional nervous disorder.

She was still able to do a little sweeping and washing up at times, but had hardly left her home for some years. She had noticed that her eyelids would droop at times so that the eyes would almost close. At times, too, she may have seen double slightly. She was well nourished and

well developed. She had slight ptosis, but no ophthalmoplegia (Figure VII). There was gross weakness of the arms, and her grip was not measurable with the dynamometer. Her legs were fairly strong, but she was unable to sit up from the lying position unaided.

Following the injection of 1·25 milligrammes of "Prostigmin" the ptosis disappeared, the arm movements became strong, and the hand grip two-thirds that of an average normal woman (Figure VIII). She stated that she felt perfectly well, which she had not felt for many years. She has since had four hours of each day in which she can go out and enjoy herself after the injection of a small dose of "Prostigmin". This is all she is able to afford at present.

Group III: Doubtful Cases.

In the third group are included two cases in which the diagnosis of myasthenia was considered possible before the administration of "Prostigmin", and two cases of nuclear ophthalmoplegia. In no case was the slightest improvement observed as a result of "Prostigmin" therapy. It is therefore considered that the diagnosis of the first two cases was probably incorrect. In the second two the mechanism of the paralysis of the ocular muscles was either different from that of true *myasthenia gravis* or the muscles were too atrophic to respond. This observation is of interest, in that a comparable condition of paresis of these muscles may terminate after several years in what is apparently true *myasthenia gravis*.[5] The pathological basis of nuclear ophthalmoplegia is not as yet known.

Other patients with muscular disorders, such as muscular dystrophies, have been given "Prostigmin" without the slightest demonstrable improvement.

CASE VI.—A woman, aged thirty-five years, had been for many years suspected of having mild *myasthenia gravis*. She complained of constant muscular weakness and over-fatigability. She constantly had a mild degree of ptosis, but had never noticed any double vision nor had any great improvement in her condition followed rest (Figure IX).

She was given 2·5 milligrammes of "Prostigmin" hypodermically. Although she experienced abdominal pain, nausea and sweating, no raising of the eyelids was observed, nor any decrease of her general weakness experienced.

CASE VII.—A woman, aged about thirty-five years, had suffered from double vision, ptosis and some difficulty in swallowing some months previously. She had since improved somewhat. She was given 1·25 milligrammes of "Prostigmin" hypodermically without symptomatic or objective benefit. She has since made a perfect recovery.

CASE VIII.—A woman, aged forty-eight years, had suffered from extreme ophthalmoplegia, affecting all the external eye muscles for nine years. No movement of the eyes could at any time be observed. Otherwise she was in good health and presented no neurological abnormality (Figure X). An injection of 2·0 milligrammes of "Prostigmin" had not the slightest effect upon her condition.

CASE IX.—A man, aged sixty-five years, had suddenly become affected with left facial paralysis some years previously. Subsequently he suffered from double vision and drooping of the right upper lid. He had intermittently suffered from choking attacks and regurgitation of fluids through the nose. No facial movements had returned; as his left eyelids had been sewn together owing to recurrent corneal ulceration, his left eye could not be examined. He had a right internal and external ophthalmoplegia and extreme ptosis, the eye remaining in abduction. A complete left facial paralysis was present. No other neurological abnormality was found. Two milligrammes of "Prostigmin" given hypodermically produced no effect on the movements of the eyes.

It cannot be said unequivocally that the ophthalmoplegia in the two latter cases was of other origin than that of *myasthenia gravis*. It is not impossible that the ocular muscles were so atrophic as to be unable to contract. Had either patient shown other signs by which the effects of "Prostigmin" could have been judged, this point might have been made clear. It would seem, however, that in a long-standing case of nuclear ophthalmoplegia the patient is unlikely to obtain benefit from "Prostigmin" therapy.

FIGURE IX.

Case VI: Patient suffering from a condition resembling *myasthenia gravis* not relieved by "Prostigmin".

FIGURE X.

Case VIII: Patient suffering from external ophthalmoplegia not relieved by "Prostigmin".

Conclusions.

The hypodermic injection of "Prostigmin" offers a dramatic means for the immediate improvement of the symptoms of *myasthenia gravis*. As this drug is freely absorbed from the gastro-intestinal tract, it is likely that the oral method of administration will eventually be largely used. As the useful oral dose requires to be as much as 25 to 30 milligrammes, it is obvious that only a small percentage of Australian patients could until recently afford to use it other than hypodermically. It is likely that certain patients could be kept in an almost constant state of well-being were they able to take "Prostigmin" orally two or three times a day.[4][6] Patients of group I and group II have recently been treated by the oral administration of "Prostigmin" with great benefit.

In this series five patients with true *myasthenia gravis* responded well to the injection of "Prostigmin"; two patients with a disorder resembling *myasthenia gravis* did not do so. It is possible that these cases were not true *myasthenia gravis* or that a type exists in which the chemical anomaly, which presumably leads to the non-transmission of the nervous impulse, is of a different order. Two patients with nuclear ophthalmoplegia failed to respond. It may have been that the ophthalmoplegia was of different origin from that of true *myasthenia gravis*; on the other hand, it is possible that too

great a degree of atrophy and fibrosis had taken place in the disused ocular muscles to permit of contraction, even when the nervous impulse was able to pass.

Acknowledgements.

I am greatly indebted to Dr. A. B. Corkill for the interest he has shown in *myasthenia gravis* and the biochemical studies that he has undertaken. My best thanks are due to my colleagues who have referred to me patients suspected of suffering from *myasthenia gravis*.

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THE COPPER SULPHATE TREATMENT OF TROPICAL ULCER AND NEW GUINEA MOUTH DISEASE.

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TROPICAL ULCER and New Guinea mouth disease differ only as to the site of occurrence; each is fundamentally due to food deficiency, each presents as a foul, sloughing ulcer, from each can be isolated a spirochæte and a fusiform bacillus, and each responds readily to the same form of treatment. Both are almost entirely confined to natives.

Tropical Ulcer.

The first type of tropical ulcer occurs as a large sloughing ulcer on the dorsum of the foot, on the ankle, or on the lower two-thirds of the leg. Following even slight trauma, such as a superficial abrasion, the area around the injury becomes tensely swollen for a radius of 1·25 to 5·0 centimetres (one-half to two inches), appearing as a blunt conical swelling with its apex at the site of injury. There is no discharge, nor is there pus when it is incised. This stage develops in from one to three days, and is apparently one of invasion by the specific organisms, with thrombosis of all vessels, including those of the skin as far out as the margin of the swollen area.

The next stage is one of rapid sloughing of all this area. The process is astonishingly fast—often the transition occurs overnight, from a conical swelling to a four-inch ulcer with steep, sharply defined edges, filled with a stinking, soft, shaggy, grey-green slough; this slough is firmly attached to the base. Usually neither bone, tendon nor muscle is involved; the process extends only through the skin and subcutaneous tissues.

When copper sulphate is used there are no relapses; but otherwise it is typical for an ulcer which is apparently clean and healing well, even at the stage in which the skin is advancing over the granulation tissue, to break down overnight, so that it is as large as, or larger than, the original ulcer.

Here fitting tribute might be paid to one Muru, a hospital orderly. One of his dressings was in a case of tropical ulcer in which two relapses had previously occurred. Entirely on his own initiative he cauterized the granulations over about one-third of the area daily with a crystal of copper sulphate. When the inevitable relapse occurred, the cauterized area did not share in the sloughing; the writer took the hint, experimented with copper sulphate, and the ulcer healed rapidly.

Muru was later stationed in an isolated spot where fresh vegetables were scarce; he was brought in from there with well-developed New Guinea mouth disease. *Liquor Arsenicalis* and *Vinum Ipecacuanhæ* were tried without effect. The striking resemblance of the ulcer to a tropical ulcer suggested a trial of copper sulphate; but although there was immediate improvement in most areas, the ulcer persisted behind the last molar, and ultimately eroded into a vessel, possibly the internal maxillary artery or one of its branches. Muru was found dead early one morning.

The second type of tropical ulcer occurs in the distal half of the terminal phalanges of the fingers and toes. It commences as a swelling around the nail, after which the tip of the finger rapidly becomes bulbous and then sloughs. When the slough is detached, the base of the nail remains firmly attached; but the skin and soft tissues as far back as this are missing, and the tip of the phalanx is seen projecting from the base of the ulcer below the nail.

In both types, once sloughing has occurred and tension is relaxed, the condition is practically painless.

New Guinea Mouth Disease.

The preliminary stages of New Guinea mouth disease are on the gums, but the final stage involves the cheek. There is first what might be called a "marginal gingivitis", extending alongside a varying number of teeth. The gums rapidly swell and ulcerate along the margins, and the process extends into the tooth sockets, so that the teeth project slightly and are very loose. Frequently the alveolar margins of the jaw bones are involved, and sequestra form.

Once ulceration of the gums is established, the process is transferred to the contiguous areas on the inside of the cheek. Here there forms a sloughing ulcer of from 1·25 to 3·75 centimetres (one-half to one and one-half inches) in diameter, identical in appearance with the typical tropical ulcer, with its base formed by the skin of the cheek. Externally there appears a dusky area of skin, corresponding in extent with the ulcer within. Shortly this area of skin sloughs, leaving a sharply-cut hole through the entire thickness of the cheek.

Occasionally the ulcer occurs far back on the medial side of the ramus of the mandible, which forms part of the floor of the ulcer. Here there is danger of erosion of vessels. Occasionally also there may be ulceration of the soft tissues of the hard palate, or perforation of the soft palate; the uvula may slough, or the tonsils may ulcerate. The gingivitis is painful, and pressure cannot be brought to bear on loosened teeth, but the ulcers on the cheeks and palate are almost entirely painless.

Among indentured labourers on a liberal diet, including abundance of fresh vegetables, both of these conditions are rare; when vegetables are scarce and diet is confined almost entirely to rice and tinned meat, they are common.

General Treatment.

The first essential in general treatment is a diet rich in vitamins. The patients are given no rice, but receive abundance of fresh vegetables; "Marmite" and cod liver oil are added. Rest is essential.

Local Treatment.

In local treatment copper sulphate acts with such rapidity in comparison with other forms of treatment that it can be regarded as specific. It is best used as a solution of 1 in 150 approximately; for practical purposes this amounts to a level teaspoonful of the crystals to the standard New Guinea medicine bottle of 26 ounces capacity. It is applied as a wet compress to ulcers, and employed as a mouth wash in New Guinea mouth disease and as a continuous bath for the second type of tropical ulcer.

Local Treatment in Tropical Ulcer.

First Type.—The compresses are of lint soaked in the solution, applied without being wrung out, and lightly bandaged. These are renewed every four hours until the slough separates; once this has occurred they are changed only night and morning, but in the intervals the compress is moistened with the solution whenever it becomes dry. This is continued until the ulcer has almost filled with healthy granulation tissue, when scarlet red ointment and adhesive strapping are applied. The granulation tissue is always covered by a greenish-black layer which can easily be removed, although removal is not necessary.

Under this regime the slough will separate within thirty-six hours, leaving a firm, clean floor. Within a further ten days the ulcer will have filled up sufficiently for it to be strapped. Further time involved depends entirely on the area to be covered with skin. If this is very extensive, grafts may be employed; it is advisable first to prepare the skin which is to be used for grafting by applying copper sulphate compresses for two days. Exuberant granulations should be touched with a moistened crystal of copper sulphate.

A fragile, non-pigmented scar usually results, and such patients should be drafted to occupations in which they will be less likely to suffer minor trauma.

Second Type.—In the second type it is futile to expect that the tip of the digit will reform; hence copper sulphate is only of use for preliminary cleaning up. It is employed as a continuous bath all day, and a compress is applied for the night. The slough separates within thirty-six hours, and in two or three days the base of the ulcer is covered with healthy granulation tissue. Amputation is then performed, usually through the distal interphalangeal joint, although sometimes the available flaps are inadequate and it is necessary to perform amputation through the shaft of the second phalanx. Local anæsthesia with "Novocain" and adrenaline is sufficient. No elaborate skin preparation is required, as the continuous bath appears to render the skin sufficiently clean; it is enough to swab the finger with ether before commencing the injections. In dealing with natives it is advisable to flex the remaining fingers during the operation and to bandage them down to prevent interference. On general principles catgut should be avoided if possible; the skin sutures of silkworm gut can be planned so as to act as hæmostatic sutures. Healing occurs by first intention.

Local Treatment in New Guinea Mouth Disease.

Most patients with New Guinea mouth disease come under treatment before the cheek is involved. In this stage a 26-ounce bottle of solution is issued each morning, and the patient is instructed to use it frequently as a mouth wash or gargle, holding it in the mouth for as long as is comfortable. This quantity should be sufficient for twenty-four hours. There will be marked improvement after twelve hours, and complete healing within four days.

When the teeth are involved, closing the mouth is usually painful, and the mouth should be syringed out with the solution at intervals. It is sometimes possible to lay a strip of lint over the affected teeth to obtain more continued application of the solution; the strip should be wide enough to fall down over the gums on each side, and should be moistened frequently. In no circumstances should the teeth be extracted, no matter how loose they appear; nor should the patient be allowed to remove them with his fingers. This is vital, as extraction opens up a large field for absorption, and the patient invariably dies. When the condition has entirely settled down the teeth may reset themselves firmly, otherwise they may be extracted. Sequestra should be removed only after the condition has entirely settled down; they are usually black, wedge-shaped and loosely held by the adjoining mucous membrane, which extends over the edges of the sound bone.

When the cheek has ulcerated, until actual perforation occurs the solution may be used as a mouth wash. This sometimes prevents final perforation. After perforation has occurred the solution should be used with a syringe. If they will remain in place, pledgets of cotton wool may be packed between the gums and cheek. The edges of the perforation should be rubbed thrice daily with a moistened crystal of copper sulphate. Even moderately large perfora-

tions will fill in under this treatment. In extreme cases plastic operation may be necessary after the condition has settled down.

When ulceration occurs far back, behind the third molars, the case is fairly hopeless. The frequent application of powdered copper sulphate on a damp wool swab may be of value. Death is usually due to hæmorrhage from an eroded vessel.

Other Effects of Copper Sulphate.

There appear to be no ill-effects from the use of copper sulphate as detailed here. The teeth are not discoloured, nor do they subsequently show signs of decay. Healthy mucous membrane is unaffected. The skin around the ulcers is not damaged; it does not even become sodden. Natives, at any rate, do not object to the taste. There must be considerable absorption of copper sulphate through the ulcerated surfaces, but no constitutional effects have been noted. Even when a patient, misunderstanding his instructions, swallows a few mouthfuls of his mouth wash, nothing has been noted beyond occasional immediate vomiting.

Conclusions.

1. Copper sulphate, in a dilution of 1 in 150, is apparently specific for tropical ulcer and New Guinea mouth disease.

2. Its use shortens the duration of these diseases, stimulates healing and prevents relapses.

3. No ill-effects have been observed from its use.

4. It is cheap, simply prepared, and has the advantage that the crystals are easily transported; there is no need to carry fragile bottles of fluid, a consideration where all carrying is done by native porters. For mixing and issuing the solution empty 26-ounce bottles are always available in abundance throughout the tropics.

Reviews.

RECENT WORK ON PULMONARY TUBERCULOSIS.

THAT "Recent Advances in Pulmonary Tuberculosis" by Burrell has reached its third edition is some indication of its value.[1] The book is written more for the help and guidance of the general practitioner and student than for the specialist in tuberculosis, and provides a useful collection of information and theories, together with expressions of opinion by the author on questions which are open to debate.

Big advances have been made in the surgical treatment of plumonary tuberculosis during the last few years, owing in some measure to the greater degree of efficiency in radiography and in the administration of suitable anæsthetics. These advances are discussed at some length, and new chapters dealing with infectivity and immunity, and with bovine and childhood tuberculosis are included.

Statistical tables are inserated freely, and illustrative case records are numerous. Chapters are arranged in good sequence and the frequent references are tabulated at the end of each chapter, while a scheme that makes an appeal is the collection of skiagrams at the end of the book. The type and paper leave little to be desired; only two typographical errors were detected.

Several paragraphs were encountered which left the impression that they could have been more lucidly written, but generally speaking the book is easily readable. However, after reading it one is inclined to feel that the forms of treatment discussed are not of much avail. One by one they are stood up, evidence by various authorities are adduced in their favour or otherwise, and finally they are more or less skittled by the author. Nature, assisted by the discipline and routine of a sanatorium, collapse therapy whether by artificial pneumothorax or some form of thoracoplasty, and to a lesser extent the injection of aqueous solutions of gold salts seem to withstand this skittling process best. Tuberculin is dealt with by quoting some of the experiences and recommendations of various workers, but reference to the work of Camac Wilkinson is conspicuous by its absence, and Gillespie gets hardly more than an honourable mention. The author's oft repeated suggestion that shock plays the main part in the therapeutic use of tuberculin, and its frequent comparison with the injection of "TAB" will not appeal to those whose technique has enabled them to obtain consistently good results with tuberculin.

On page 42 the last line reads: "Since 1 c.cm of O.T. contains 1,000 mg" *et cetera*. One wonders whether the author means to suggest that one cubic centimetre of "O.T." weighs 1,000 milligrammes, or is he seriously stating that it contains 1,000 milligrammes of something: if so, what is it? In any case, why invoke the aid of milligrammes when "O.T." is essentially a fluid? Again, dealing with the von Pirquet test, the author states: "This test is equivalent to 1 in 10,000 dilution by the Mantoux method." To justify this assertion one would have to take care in scratching the arm that exactly 0·0004 cubic centimetre of the drop of fluid used became effective. It is a pity to mar an otherwise good and useful book with such errors.

In conclusion, one can confidently recommend the book to those who desire in a concise form modern views on many aspects of tuberculosis.

[1] "Recent Advances in Pulmonary Tuberculosis", by L. S. T. Burrell, M.A., M.D., F.R.C.P.; Third Edition; 1937. London: J. and A. Churchill Limited. Large crown 8vo, pp. 323, with 48 plates and 22 text figures. Price 15s. net.

THE PATIENT AND THE WEATHER.

DR. PETERSEN has published Part I of the fourth volume of the series "The Patient and the Weather".[1] He states that this volume is intended primarily to demonstrate that organic disease may take its origin from subminimal environmental changes. A number of case histories are presented concerning patients suffering from respiratory, renal, cardiac, vascular and blood diseases. The major incidents in these histories are charted in relation to coincidental meteorological occurrences. Frequently more complicated charts are given which combine the results of numerous clinical and biochemical investigations. Dr. Petersen claims in almost every case that the meteorological changes are significant in ætiology. He emphasizes particularly the importance of major and minor polar infalls. But he is satisfied when analogous clinical events occur before, during or after a polar infall. His postulates, therefore, concerning the effects of different phases of changes in the weather tend to drift into plausibilities. Moreover, he gives no assurance that his laboratory findings were obtained under standard conditions in regard to time of day, the taking of food *et cetera*. Occasionally data concerning more than one patient are recorded in relation to identical weather conditions. An adequate number of detailed investigations of this type would be most welcome and should give results of unquestionable value. Dr. A. J. Nedzel has contributed in a final chapter an advance report on experimental endocarditis in dogs subjected to the pressor effects of pituitrin.

[1] "The Patient and the Weather", by W. F. Petersen, M.D., with the assistance of M. E. Milliken, S.M.; Volume IV, Part I: Organic Disease—Cardio, Vascular, Renal Disease, including a chapter on experimental endocarditis by A. J. Nedzel, M.D.; 1937. Michigan: Edwards Brothers, Incorporated. Imperial 8vo, pp. 699, with illustrations. Price: $10.00 net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 19, 1938.

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THE CONTROL OF HUMAN TUBERCULOSIS.

SOME of the methods suggested for the control of tuberculosis in a community must be regarded as rather Utopian. For example, we are told that the Mantoux test should be applied to the whole population as a routine measure; that those individuals found to be uninfected should be retested at stated periods; that frequent clinical, radiological and pathological reexaminations of each infected patient should be made; and that all tuberculous patients should be segregated while they are infective. Chester A. Stewart, in an article on the subject, rightly calls this an idealistic but expensive programme; at the same time he points out that it is but the parallel in the human population of the course already successfully adopted in the United States of America in connexion with bovine tuberculosis.[1] It is perhaps better to refrain from unfair comparisons between the practice of prophylaxis in veterinary and human medicine. But Stewart has evolved a plan, much more modest, but practical and probably quite effective, by which household units are surveyed, and, as he puts it, accredited if they are free from infection. His idea is that a large number of private practitioners should be utilized to promote an unsubsidized tuberculosis survey by means of the Mantoux test. The results of this relatively inexpensive investigation would enable the examiners to determine whether further investigation was necessary. Thus if parents were found to be tuberculin-sensitive and their children showed no reaction to the skin test, it could be assumed at least that the parents were not passing on a tuberculous infection to their children, and therefore the more expensive examinations of the adults could be dispensed with on that occasion. Families of this type, as well as those none of whose members gave evidence of infection, could be classed as safe, that is, free of contagion. Of course, should the number of infected persons in the house increase, this tentative rating would need to be reviewed. The practical interest taken in New South Wales at the present time in connexion with a specially subsidized Mantoux survey gives local point to Stewart's suggestions. To be effective such a survey would need the cooperation of a large number of medical practitioners, for the task of controlling or even tracing the dissemination of human tuberculosis is colossal. One practical drawback would be in obtaining the cooperation of the household units themselves, especially among the poorer or more careless member of the community; and it will be evident, too, that the poorer a community, the more patients must a doctor see in order to make a living, having thus but little time left for even so simple a procedure as performing a series of intracutaneous tests on a large number of people.

Stewart quotes some results. During a three-year period an intradermal injection of 0·1 milligramme of old tuberculin was administered to 1,593 persons belonging to 642 households. Only in one-third of the families was the survey complete, usually because the father was not tested. Only 5% of the children were found to be sensitive to tuberculin at the age of six years. Analysis of the figures appears to indicate, however, that the primary stage of tuberculous infection does not seem to be an important source of childhood infection. One case history quoted in the text illustrates the great difficulty in tracing infection by a statistical survey. One child was found to be tuberculin-sensitive in a

[1] *American Journal of Diseases of Children*, October, 1937.

family otherwise uninfected, though the father was not tested. The lack of infection in the other children seemed to exonerate the father, and this assumption is apparently a just one, for it was found that the child had been in contact with a tuberculous aunt who was awaiting admission to a sanatorium. Radiological study revealed no abnormality in this child's chest, yet five years later she had an hæmoptysis and a cavity was then found in one subapical region. The value of the Mantoux test in pointing the finger of suspicion at an infected person is strikingly illustrated in this instance. The tables and discussions in this paper are too detailed to be of value here, but surely we must agree that the economy of the method described makes it particularly suitable for private practice. The general practitioner now adds immunization to his activities; why should he not go a step further and undertake an antituberculosis service for the families under his care? Obviously he cannot cry his wares in the market place, but it might be possible when the suspicion of tuberculosis arises, as it so often does in families, to suggest this simple and useful method of investigation. Stewart claims that merely by the application of Mantoux tests nearly 90% of the completely tested household units were "accredited" or tentatively rated as safe for children to live in. Unfortunately the lengthy and unsatisfactory nature of pulmonary tuberculosis as an illness has made it extremely unpopular with general practitioners, who often feel that they have, unhappily, not much useful work to do for the luckless sufferer from advanced disease. But a scheme such as this is entirely different; it would enable the disease to be detected earlier and in numbers of cases to be prevented. The whole idea is worth more careful thought by those interested in preventive medicine.

Current Comment.

FLUID ADMINISTRATION AFTER OPERATION.

THE question of fluid administration after operation has recently been reviewed by W. W. Walther,[1] who has made a study of the water balance of fifty patients following surgical operation. Twelve such patients received no treatment. In twenty-seven instances the surgeon considered it advisable to order fluid by rectal infusion or to "force fluids" by mouth. A further nine patients received an intravenous infusion of saline solution. Estimations of the relative concentration of red cells in the plasma were performed regularly by the Haldane hæmoglobinometer and the hæmatocrit, and readings of plasma chloride and plasma protein by approved methods were simultaneously recorded.

The results were both interesting and instructive. Patients who received no excess of fluid after operation, or who were even restricted in the fluid intake, showed a slow reduction of hæmoglobin percentage after operative hæmorrhage, and the percentage often took some days to reach its new level. This means that the normal process of dilution took a long time. The patients were often partially dehydrated by this time. Even allowing for the usual graver condition of the group who received abundant fluids by mouth or rectum, these patients recovered more quickly. Their blood dilution was soon obvious. No important reduction occurred in the chlorides. Patients treated by large quantities of saline solution given by the rectum developed hyperchloræmia. Protein followed much the same fate as hæmoglobin. Half the patients untreated by fluids manifested a loss of plasma protein; the majority of the "treated" patients lost none. Four patients developed pulmonary œdema, while the plasma protein was below the normal level of 5·8%. In three of the four patients thus affected, the condition developed after the intravenous administration of saline solution.

The biochemical picture seen in shock was most advanced in patients who were untreated post-operatively by fluids. This picture consists of a concentration of corpuscles in peripheral capillaries, and a loss of plasma protein beyond that due to hæmorrhage.

As preventives of shock, therefore, there seems little to choose between fluid given *per os* and fluid given *per rectum*, but an excess of salt may cause hydræmia and predispose to pulmonary œdema. The intravenous route, though used so frequently, is, it seems, not the best. Water is absorbed best when given through the mouth. Even in small quantities it is of greater value than a greater volume placed under the skin or directly into the veins. It has to pass through the portal system and probably picks up protein *en route*. The best nurse, then, is she who can persuade the patient to take sips of water at frequent intervals. The chief risk of the intravenous administration of fluid is the risk of pulmonary œdema by the reduction of osmotic pressure. If urgent restitution of blood volume is required, it is better to give one containing protein, that is, plasma itself. Red cells are not called for at this stage, and it is unnecessary to impose a polycythæmia upon shock.

So the pendulum begins its return journey. We are now told that the best method of fluid replacement, if the oral route is contraindicated, is by the

[1] *The Lancet*, January 2, 1938.

rectal drip method. The rectal administration of over two pints a day considerably shortens the time for blood dilution. Since no evidence of serious chloride deficiency is found after operation, the fluid need not contain salt at the concentration found in "normal" saline solution. Determination of the level of plasma protein should ideally precede the intravenous administration of fluid. If this level is low, further dilution may encourage the development of pulmonary œdema. In such conditions as severe burns, for example, in which considerable protein loss and blood concentration occur, it would appear that emergency treatment should consist in intravenous infusion of plasma rather than blood transfusion. The latter procedure carries a risk of increasing the already high concentration of red cells in the blood and so increasing stasis.

CORONARY DISEASE IN YOUTH.

WHAT amounts to a new angle on the study of longevity has been attempted by the Bostonian cardiologists R. Earle Glendy, Samuel A. Levine and Paul D. White.[1] They have assembled data on the mode of life of 300 persons living beyond the age of eighty years, the assumption being that such persons represent a selected group of individuals in whom the development of arterial degeneration, and in particular coronary sclerosis, was delayed as a result of the non-operation of certain recognized "ageing" processes. With greater difficulty these authors have also accumulated facts concerning 100 persons in whom evidence of coronary disease made itself apparent before the age of forty, and who may be assumed to represent the converse, a selected group in which degenerative processes have been unduly accelerated.

This novel and most interesting comparison represents the result of some four years' labour. Each patient was admitted into the inquiry only if coronary thrombosis had occurred, or if *angina pectoris* was present, or in the absence of any other factor which might induce cardiac pain, or if they showed electrocardiographic evidence of coronary disease. In this way only uncomplicated instances of coronary disease were accepted. Information was obtained by an extensive *questionnaire* relating to birthplace, racial stock, ancestral longevity, residence, occupation, exercise, diet, use of tobacco and alcohol, and past history of infections. The scope of a statistical comparison of these factors can be well imagined, and the original report must have been a voluminous one. The main deductions, however, can be crystallized as follows.

Relatively far more (90%) of the old group than of the young group were of British stock. Only 44% of the young group were of British stock, while 39% were Jewish. This is a further confirmation of the unusually high vascular vulnerability of this race. It must not be forgotten, however, that the proportion of Jews in eastern America is relatively high, and that no mention is made of a possibly high Jewish influx into Boston in the forty years' interval separating the averages of the groups of persons under consideration. As regards the influence of ancestral longevity, it appears that the average age at death of the fathers of the octogenarians exceeded by nine years the figures of the young group, and of the mothers, by fourteen years. The majority of the old group resided in rural districts, whereas almost all of the younger persons had led urban existences. There were more professional and business men in the older group. As regards the amount of exercise which had been indulged in, a considerable difference was apparent. Practically all members of the old group had taken exercise to well beyond middle life, whereas half of the younger group were of strictly sedentary habits and took very little exercise. No conclusions of value could be drawn as regards the respective diet of the two classes. The figures as regards tobacco consumption are interesting. Of the older group, 55% were smokers, whereas 93% of the young group indulged in tobacco. As regards alcohol consumption, no significant difference was apparent. It was surprising to note that the older group had suffered more severely from infection than the more recent generation. It would appear, therefore, that infections do not have an important rôle in the early production of coronary disease. The characteristic habitus of most of the old group in early life was thin or lean, whereas most of the younger group were robust in stature. One-third were definitely obese. Nervous sensitiveness and strain were considerably greater in the younger group and practically negligible in the older group. We cannot accord much importance to such a comparison, however, since occupation, race and a greater speed of life must all be taken into consideration.

The following facts as regards the characters of coronary disease in early life also emerged from this study. Approximately 1·7% of all coronary disease occurs in persons under forty; these are overwhelmingly the victims. Hypertension as an important factor is predominant in women. A greater number of young patients than patients in general may be expected to have hearts that are normal in size. There are fewer complications, and diabetes or evident peripheral vascular disease is uncommon. The duration of life for those who died and the life expectancy of the survivors are greater than for patients of all ages with coronary disease, but the susceptibility is just as great. Inheritance and ancestral longevity are as important factors in the early as in the late occurrence of coronary disease.

This study has been most painstakingly concluded. It gives statistical support for much that was already strongly suspected. The information concerning the prognosis for the younger sufferers from coronary artery disease is more vitally necessary than that which concerns the senile. There is much material here of use to health and "keep fit" propagandists.

[1] *The Journal of the American Medical Association*, November 27, 1937.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Investigations into the Transit of Ova in Woman.

AXEL WESTMAN (*The Journal of Obstetrics and Gynæcology of the British Empire*, October, 1937) reports the results of investigations that he has made into the transit of ova in woman. After a review of the literature back to 1858, he summarizes experimental work that he carried out on monkeys and reported in previous papers. In his investigations of the problem in the human being, he took steps when operating to inject a drop of lipiodol under the *tunica albuginea* of the ovary at the upper and lower poles so that the organ could be visualized by X rays at a later date. Nine cases were studied by the author. He found that in seven of them there was considerable movement of the ovary, a condition that he had proved to occur in monkeys. The ligaments of the uterus, tubes and ovaries contain much smooth muscle tissue. Therefore the tubes and ovaries are capable of considerable movement. In conjunction with the injection into the ovaries, utero-salpingography was carried out to verify the anatomical relationship of the ovary and tube. Serial photographs were taken in several cases. Some of these photographs show that the ovaries move cranially and caudally as well as laterally and medially. The ovary may also rotate on its axis, and the tube may curve round the ovary in a bow-shaped manner. The author has come to the conclusion that, on account of this mobility, the infundibulum at the time of ovulation can be placed in direct contact with the ovary, which, through rotary movements, has the power of turning its various surfaces towards the *ostium abdominale tubæ*. In such circumstances the ova liberated from the ovary undoubtedly will never enter the general abdominal cavity, but will be directly transferred from the ruptured follicle to the Fallopian tube.

Granulosa-Cell Tumours of the Ovary.

FREDA BURY PRATT (*The Journal of Obstetrics and Gynæcology of the British Empire*, October, 1937) has undertaken a review of the literature of granulosa-cell tumours of the ovary, which were first described in 1895 by von Kahlden as adenoma of the Graafian follicle with transition to malignancy. The author gives a complete summary of the literature to date with a description of the morbid anatomy, histology, signs, symptoms and treatment of the conditions. Typical granulosa-cell tumours of the ovary are unilateral, soft, solid, or partly solid and partly cystic, encapsulated and vascular. The surface on section is pinkish or fleshy in colour or bright yellow. They fall into three main histological groups, with transitional forms between these: (i) folliculoid, (ii) diffuse or sarcomatoid, (iii) cylindroid or trabecular. In addition, the *oophoroma folliculare*, or Brenner tumour, is probably a variety of granulosa-cell tumour. In all of them the component cells resemble follicle cells at various stages, from primordial follicle to mature granulosa about to be luteinized. The uterus in cases of granulosa-cell tumour is enlarged to the size of a uterus that is pregnant two to three months, and there is cystic hyperplasia of the endometrium. Such after-histories as are available indicate that the majority of typical granulosa-cell tumours are non-malignant, though the histological picture often suggests malignancy. Some malignant tumours described as granulosa-cell tumours may be misdiagnosed as such. They may occur at any age of life. The greatest incidence and liability to these tumours is between the ages of forty and sixty years, that is, about the menopause. The greatest incidence of the Brenner tumour is between sixty and seventy years of age. In childhood they cause precocious puberty with uterine bleeding. In active adult sexual life they cause menstrual irregularity, or amenorrhœa followed by uterine bleeding, which may be regular or irregular and is usually excessive. After the menopause they cause post-menopausal uterine bleeding, which may be regular, resembling normal menstruation. The tumours probably produce large quantities of œstrin. This is thought to be the cause of the hyperplasia of the uterine endometrium. The symptoms, other than mechanical ones due to the presence of an ovarian tumour, probably depend on this production of œstrin. Treatment is unilateral salpingo-oophorectomy on the affected side, more radical removal being necessary only if clinical evidence of malignancy is obvious at the time of operation. The prognosis is good, and this treatment promises return of the patient to normal in every way. Post-operative radiotherapy is not indicated.

The Effect of Pregnancy on Malignant Tumours.

F. R. SMITH (*American Journal of Obstetrics and Gynecology*, October, 1937) reviews the histories of 54 patients and is of the opinion that pregnancy is detrimental to and should be prevented in patients suffering from unarrested and malignant tumours. Although temporarily retarded by pregnancy, the growth of such tumours is accelerated after the termination of pregnancy. If pregnancy is interrupted, prognosis is not so good. The prognosis is better if in tumours of the breast and non-genital tumours the pregnancy is not terminated regardless of time relationship of pregnancy to the occurrence of the tumour. It is a distinct advantage to the patient to treat the tumour and ignore pregnancy, excepting in melanomata, which are especially apt to spread when stirred up. Irradiation of the breast has no tendency to produce malformation of the baby, and the author states that in the latter months of pregnancy irradiation can be employed for carcinoma of the cervix without affecting the baby or producing abortion.

Appendicitis and Menstruation.

K. HOLLÓSI (*Monatsschrift für Geburtshülfe und Gynäkologie*, September, 1937) discusses the possible relationship between appendicitis and the menstrual cycle. He recalls that the pre-menstrual swelling of the genital tract is due to the luteal hormone, and suggests that such hyperæmia is not confined to the pelvis, but also involves the intestinal tract. In a series of 159 cases of appendicitis the characteristic signs were noted in the pre-menstrual and post-menstrual periods in two-thirds of the cases. Frequently at operation the appendix was observed to be swollen and covered with small hæmorrhages. While the diagnosis may not be easy, a decision can usually be reached after careful observation of the patient. He draws attention also to the connexion between dysmenorrhœa and chronic appendicitis in many cases. On the removal of the appendix many such patients with chronic dysmenorrhœa are cured.

OBSTETRICS.

Analgesia, Anæsthesia and the New-Born Infant.

S. H. CLIFFORD AND F. C. IRVING (*Surgery, Gynecology and Obstetrics*, July, 1937) criticize modern methods of obstetrical analgesia in relation to their efficiency and their effects on the new-born infant. Their investigation covers analgesia by multiple methods as well as by single agents, and they include only vertex presentations in their series of cases. They issue a warning against the use of any analgesic containing opium or its derivatives, and state that the use of morphine within four hours of the birth of a premature infant will double the mortality rate. They offer evidence that the pentobarbital-"Amytal" drugs are more than twice as efficient as morphine when used as analgesic agents. When barbiturates were used, only 3% of babies needed any form of resuscitation at birth, whereas at least 23% of those in the morphine series required active treatment. Again attacking the use of morphine they state that the condition of the infant at birth is influenced both by the dose given and by the time interval elapsing between medication

and delivery. The death rate is increased in proportion to the size of the maternal dose, and this effect may be seen even if the drug is given at a time as remote as eight hours before the actual birth. Scopolamine used in conjunction with the pentobarbital-"Amytal" group is said to produce a more complete maternal amnesia. On the other hand, the use of ether or paraldehyde by the rectum, together with the barbiturate compounds, did not result in an analgesia any more successful than was given by the barbiturate alone. "Pernocton" is dismissed as inferior in analgesic properties to pentobarbital or "Amytal", and more abnormal cyanosis of the infants was encountered after its administration. Nitrous oxide and oxygen mixture is favourably reviewed in regard to its effect on the babies; but the proportion of nitrous oxide to oxygen should not exceed 85 : 15. The authors prefer pentobarbital (in an average dose of 0·36 gramme) to "Amytal" as being safer for the mother. They stress the fact that efficient analgesia must be followed by the birth of a sleepy baby. Their method, however, has not produced any rise in the still-birth or neonatal mortality rate over a five-year period at the Boston Lying-In Hospital, and they claim 84% maternal amnesia.

Physiology of Lactation.

A. G. KOCHS (*Monatsschrift für Geburtshülfe und Gynäkologie*, August, 1937) gives the results of his experiments regarding the endocrine control of lactation. The luteal hormone acts on the main milk ducts, causing hypertrophy and enlargement, but has no effect on the more specialized elements of the mammary gland, nor can it initiate lactation. The follicular hormone, on the contrary, stimulates the finer tubules and alveoli, and when in excess causes the development of an atypical parenchymatous growth. The two ovarian hormones act in unison, as in the preparation of the endometrium for the embedding of the ovum; but in the breast the first phase is controlled by the luteal hormone, which needs to be followed by the follicular hormone to produce the final result. After this preliminary stimulation the effect of the lactogenic hormone of the anterior lobe of the pituitary is still necessary. This hormone will not work in the presence of an excess of follicular hormone, therefore it is essential for successful milk secretion that the œstrin be excreted as rapidly as possible from the circulation.

Clinical Experience with a New Ergot Alkaloid.

THE use of ergobasine tartrate in the third stage of labour is reported by G. E. Tritsch and K. H. Beihm (*American Journal of Obstetrics and Gynecology*, October, 1937). The drug was given to 115 patients, of whom 36 were *primiparæ* and 79 *multiparæ*. The authors came to the conclusion that basergin ergobasine is a powerful oxytocic. When given intravenously at the beginning of the third stage of labour it caused uterine contractions to be resumed in an average period 22 seconds and 34 seconds in *primiparæ* and *multiparæ* respectively in their series of cases. This new ergot shortens the third stage of labour to an average of 2·95 minutes. Bleeding is lessened, the majority (80%) of patients bleeding less than the average. In four cases hour-glass contractions were met with on delivery of the placenta. In one case manual extraction was necessary.

The Use of a Rectal Bag during Labour.

W. GÜNTHER (*Monatsschrift für Geburtshülfe und Gynäkologie*, September, 1937) has investigated the use of a small rubber balloon or bag inserted into the rectum as a means of stimulating uterine contractions in cases of inertia associated with premature rupture of the membranes. The bag is of the Barnes type and contains 150 to 200 cubic centimetres of sterile water. The author presents *in extenso* the details of 360 cases and states that the best results followed the use of the bag in premature rupture of the membranes at term. Generally its use was associated with the administration of small doses of quinine. In the majority of cases contractions occurred within one or two hours and the bag was expelled within three to four hours. He did not find that the use of other means of medicinal stimulation increased the results. The only contraindication to the use of the bag was the presence of hæmorrhoids. He concludes that it is a good method of stimulating contractions and one capable of use by all engaged in the practice of domiciliary obstetrics.

The Initiation of Respiration in Asphyxia Neonatorum.

R. A. WILSON, M. A. TORREY AND KATHERINE S. JOHNSON (*Surgery, Gynecology and Obstetrics*, November, 1937) publish their opinions of various methods of resuscitation of the newborn. They deal only with the severe cases of respiratory depression and asphyxia. It is only when the oxygen content of the blood is known that a true picture of the gravity of a case can be obtained and the success or failure of the method of resuscitation properly evaluated. In severe cases of asphyxia there is a reduction in the oxygen content of the fœtal blood to extremely low levels. An important point is that although few lives are lost by the use of sedative analgesia drugs *per se*, such babies cannot stand much additional asphyxia. If obstruction or compression of the head *plus* a long deep anæsthesia takes place, these babies will die, whereas they would often recover from narcosis or asphyxia alone, but are overwhelmed when one is superimposed upon the other. The authors condemn morphine and they believe that paraldehyde is the most harmless drug. In their review of various methods of resuscitation they repeat the words of Moncrieff: "Artificial respiration in the sense of moving the chest stands condemned until respiration has begun, and once a breath has been taken it is no longer necessary." The principle of mouth-to-mouth insufflation is similar to that of the pulmotor and lungmotor; they believe that it carries a grave risk of infection. They believe that the improvement in the cardiac impulse is due to the presence of oxygen, which is absorbed from the bronchi. In their summary of this method they state that a baby is saved occasionally. Their opinion of the tilting board method is that it is of little or no value. Intubation provides a method of aspiration and an excellent airway; it should not be used in an attempt to open alveoli by direct attack. An intermittent pressure is probably of value, as rhythmic distension of the bronchial tree may bring into play the Hering-Breuer reflex. Although this reflex is absent in severe cases, it will return if the circulation improves sufficiently as a result of the absorption of the oxygen from the trachea and bronchioles. The authors believe that the best method of initiating a respiratory effort is by intravenous administration of 0·003 gramme of lobeline hydrochloride. Lobeline hydrochloride acts by lowering the threshold of the respiratory centre to carbon dioxide in the blood. Their method is to inject the lobeline into the umbilical vein, about 15 to 20 centimetres from the umbilicus, and milk the cord between the first and second fingers towards the umbilicus; this is continued slowly and progressively until a respiratory response results. They state that the intravenous resuscitation has its main object in initiating respiration. A response is usually obtained within fifteen seconds from the time the cord is milked. Their technique of resuscitation consists in a combination of intravenous medication and intratracheal insufflation.

Rupture of the Uterus.

G. VAJNA (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1937) presents the details of two cases of uterine rupture following the use of a rubber balloon to induce labour. In both instances a careful analysis of the previous obstetrical history showed that there had been some operative inteference—curettage followed by a septic puerperium. In both cases hysterectomy was performed and the specimens were carefully examined. This disclosed histological evidence of weakening of the uterine wall by scar tissue. These cases show the necessity for a careful history being taken before any method of induction involving strain on the uterine wall is adopted.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held on November 25, 1937, DR. R. E. MAGAREY, the President, in the chair.

Common Skin Diseases Met with in General Practice.

DR. L. W. LINN read a paper entitled "Common Skin Diseases Met with in General Practice" (see page 335).

DR. W. GILFILLAN read a paper in which he said that many English dermatologists still preferred to retain the term eczema, if only for the reason that so many employees associated dermatitis with compensation. Some would restrict dermatitis to that inflammation of the skin produced by a gross external irritant, such as strong sunlight or a mustard leaf, which would affect any normal skin. This was a vasomotor reaction, characterized clinically by a brisk erythema and superficial vesicles or even bullæ. Other dermatologists, Dr. Gilfillan said, would include under dermatitis those cases clinically and histologically identical with what they called eczema, but in which the history, the distribution of the eruption, or the result of the patch test suggested an external irritant as the main cause (although that irritant would not affect a normal skin); and they would reserve the term eczema for those cases in which an endogenous cause was probably the main factor. Clinically this latter type was characterized by a uniform, pin-head size eruption, which might be erythematous, papular or vesicular, which usually itched intensely, and which might go on to crusting, to scaling or to lichenification. Adamson described it as a reaction on the part of the cellular elements of the epidermis.

Discussing the ætiology, Dr. Gilfillan said that heredity was an important factor. A large number of these patients had an allergic family history, and he thought that this affected the prognosis. If it was possible to discover the cause and get the patients to avoid it, they tended in time to become sensitive to something else.

The causes could be divided into internal and external causes. Of the internal causes, the first was nervous instability; three types of eczema or dermatitis were ascribed to nervous disorder. These were nervous eczema, neuro-dermatitis or Vidal's chronic *lichen simplex*, and neuropathic eczema.

Dr. Gilfillan said that nervous eczema occurred in patients who, although nervous, were not neurotic. It was always produced by overwork or worry, especially in business men. It had all the classical signs of eczema, the itching being very severe. The usual local applications had little or no effect, and any benefit from X ray therapy was only temporary. These patients improved rapidly if they were able to take a holiday.

In neuro-dermatitis, or Vidal's chronic *lichen simplex*, Dr. Gilfillan continued, the primary factor was either itching of nervous origin or a psychoneurosis, in which the patient found it pleasant to scratch. Scratching and rubbing definitely caused the condition and kept it going. Histologically it was very similar to eczema, but there was less interstitial œdema; and although the intracellular œdema was present, rete cells and horny layer remained intact, so that blistering and weeping did not occur. But the histology differed from that of *lichen planus*, which it might resemble clinically. The affected area was bluish or brown in colour, with slight scaling; most pronounced was the thickening of the skin, which became difficult to pick up and felt tough. The fine lines of the skin were more deeply marked, and the intervening rhomboid areas projected. The edge was sharply defined.

In neuropathic eczema instability of the autonomic nervous system was present. The main feature was a vesicular erythematous eruption at the onset, becoming lichenified and mingled with pyodermia. It went on to diffuse hyperpigmentation, and ended on some areas as a vitiligo. Dr. Gilfillan added that many patients with chronic dermatoses were low acid secretors; but it was unknown whether this was the cause or the effect. Diabetes, gout, anæmia, vitamin deficiency and the old focal infection might also play an important part. With regard to focal infection, Dr. Gilfillan said that it was known that a tinea infection of the feet, for example, could produce an interdigital eczema of the hands due to an allergic hypersensitiveness of the skin to trichophytin; and the tuberculides occurring in tuberculous patients were probably of the same nature, because the tubercle bacillus could not be demonstrated in lesions or by inoculation. It therefore seemed probable that the skin might become hypersensitive to the toxins of other organisms.

Speaking of external causes, Dr. Gilfillan said that these might be light, wind, soap, friction, chemicals, dyes, dust, plants *et cetera*. These patients readily became sensitive to their own serous exudates, which not only acted as local irritants, but by reabsorption might cause fresh patches in distant parts. Another type of autosensitization had been described by Ramel in his paper on neuropathic eczema. He and other French dermatologists carried out research work to discover why a patient with dermatitis (Ramel called it eczema) due to a known irritant like cement should continue to suffer fresh outbreaks when removed from that irritant and as far as possible from all other external irritants. They proved that the new allergen might be the saprophytic microorganisms of the patient's skin, especially certain forms of yeast and certain strains of staphylococci. But Ramel stated that these could produce a reaction in the skin only when its threshold of sensitivity had already been lowered.

Dr. Gilfillan then discussed the differential diagnosis. He said that seborrhœic dermatitis had a predilection for the front of the chest and interscapular region, and was usually associated with dandruff of the scalp. The lesions were pinkish-yellow, covered with greasy scales and not raised above the surface of the skin. Streptococcal dermatitis was a superficial inflammation of the skin caused by the *Streptococcus anhæmolyticus*. It was characterized in its active stage by redness, thickening of the skin and an abundant exudation, but there were no vesicles or bullæ. The less active phase was called streptococcal pityriasis, and consisted of fine lamellar exfoliation described as being like a ploughed field. Streptococcal dermatitis attacked the folds, the retroauricular region, the axillæ, the umbilicus and the groins, and although it might become generalized, these folds were always the last and the hardest to clear up. It might occur at any age, but it was more common in children. Impetigo was caused by the *Streptococcus hæmolyticus*, Dr. Gilfillan said; the acute type presented numerous blisters, but they were more scattered than in eczema. Tinea infections, when they became eczematized, were easily and frequently diagnosed as eczema or dermatitis. Clinically there were three types: the intertriginous type, the vesicular type, and the hyperkeratotic type.

Tinea ought always to be suspected in ringed scaly lesions, especially if fine vesicles or pustules existed at the advancing edge. Where there were two opposing skin surfaces, as in the groins and the axillæ, the sharply demarcated advancing edges, sometimes with vesicles and pustules, could still be seen, but owing to moisture the central area tended to be eroded and covered with macerated horny material.

The diagnosis of eczematized or impetiginized scabies, even if this condition was kept in mind, could be difficult, Dr. Gilfillan said; but the diagnosis had already been dealt with by Dr. Linn. Dr. Gilfillan said that he had limited his remarks on the differential diagnosis in infections of the skin because in these a correct diagnosis so influenced treatment. In conclusion, Dr. Gilfillan said that it was important to suspect of being due to an infection of the skin any chronic eczematous condition limited to the external ear, to the axillæ and to the groin, and especially unilateral lesions of the hand or foot without an obvious reason for being limited to one side. The interdigital spaces of the toes should always be inspected, as they were a possible source of tinea infection.

DR. R. J. VERCO said that as diseases of the skin were responsible for quite an appreciable percentage of patients coming to a general practitioner's consulting rooms, it was to be regretted that post-graduation instruction was not available. Also, there appeared to be a considerable time-lag between the appearance of the latest information on skin diseases and its appearance in current text-books. He thought that scabies could seldom be missed if it was kept in mind, but instanced two cases in which a mistake could have arisen, one in which the condition was due to mites from tea-tree bushes, and another in which it was due to starling lice.

Dr. Verco considered that vaccines did good in the treatment of pustular types of acne. He alluded to the extreme irritability of the skin developing in some cases of acute dermatitis in which an allergic reaction existed, and quoted two cases in which good results had been obtained by discontinuing all local treatment and giving the patient injections of colloid calcium, "Ostelin" and vitamin *D*, followed by oral calcium therapy. He commented on the number of so-called regional eczemas which had been proved to be fungus infections of the skin. He asked that aural surgeons treating eczema of the external auditory meatus, which he considered usually due to fungus infections, should make inquiries as to the possibility of its presence elsewhere. He instanced a case in which fungus infection of the ear had been present for three months, but a fungus infection of the vulval and anal regions had made the patient's life a misery for twenty years.

A MEETING of the Victorian Branch of the British Medical Association was held at Ballarat on November 27, 1937, the convener of the Science Subcommittee, DR. A. E. COATES, in the chair. The afternoon meeting was held at the Ballarat Base Hospital and took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital. Parts of this report appeared in the issues of February 5 and 12, 1938.

Lingua Geographica.

DR. BASIL JONES showed a man, aged thirty-three years, who was married and in poor circumstances and had had patchy ulceration of the tongue for five years. Two years before the onset of the tongue condition he had had acute appendicitis and appendicectomy had been performed. The ulceration of the tongue was originally associated with typhoid fever, but in 1934 he had been under treatment for gastric ulcer, and he also had an injured knee with *osteitis fibrocystica*. The tongue had not shown any tendency to hæmorrhage, and no leucoplakia had been present. The condition was one of macroglossia and dryness in the mouth, which rendered eating difficult and painful. The inability to eat had caused considerable loss of weight and impoverishment of health. The gastric ulceration had been treated by means of twenty injections of "Larostidin". The blood serum had failed to react to the Wassermann test on two occasions, and short-chained streptococci had been cultured from the tongue.

DR. J. H. KELLY said that the condition was *lingua geographica*, which was more commonly seen in children than in adults. It was not a condition of ulceration, but at times was associated with hepatic ulceration near dentures and with excessive growth of fine black hairs. As it was a functional condition, and as the great dryness in the mouth was also functional, the correct treatment was to leave the tongue alone and to reassure the patient. It would gradually disappear when he ceased focusing attention on his mouth. The application of strong caustics and irradiation were to be deplored, because they were liable to produce carcinoma. The patient should have mild sedative and general hygienic treatment, with removal of depressing causes and the provision of adequate mental diversion.

DR. W. ANGUS considered that the ulceration of the stomach, which had been cured by "Larostidin", was probably also a functional state. He suggested the use of dilute hydrochloric acid in a mixture.

DR. STANLEY WILLIAMS raised the subject of subnutrition from deficiency of vitamin *C* in the diet. The patient had been unable to eat fruit and vegetables, and the diet may have been deficient, which would account for the persistence of the tongue condition and the general impoverishment of health. If it could be shown, by means of the capillary resistance test and the direct estimation of vitamin *C* in the urine, that there was a vitamin *C* deficiency, the patient would benefit by the administration of tablets of ascorbic acid.

Dr. Jones, in reply to Dr. Angus, said that the peptic ulcer had not been a pseudo-ulcer and that the patient had been taking dilute hydrochloric acid in a mixture at intervals for long periods without much benefit.

Non-Malignant Prostatic Obstruction.

DR. G. R. DAVIDSON presented the results of the surgical treatment of a series of patients with non-malignant prostatic obstruction, and referred to the methods of treatment carried out at the Ballarat and District Base Hospital. The series consisted of seventy-six patients treated by suprapubic prostatectomy, of whom four had died, and of thirty patients treated by transurethral methods, of whom five had died; of the latter five, two had appeared to die of coronary thrombosis, one after cystotomy for clot retention, and the other three had very obviously been bad risks, so common in that type of work. The majority of the suprapubic operations had been performed for the removal of adenomata, and whenever possible they had been done in one stage. In most cases it had been possible to obtain adequate bladder drainage by indwelling catheters, changed every three days and reinserted after the patient had had a bath and after the urethra had been washed out. The meatus was kept covered with a strip of gauze soaked in flavine, and the catheter was connected to tubing drained into a reservoir containing "Cyllin". Irrigations of potassium permanganate solution (one part in six thousand of water) had been given as required and followed by the instillation of silver nitrate solution (one part in three thousand of water), of which four ounces were held in the bladder for twenty minutes. Dr. Davidson said that McLean's urea concentration test had been used as the routine test of renal efficiency, but latterly the simple modification of the phenolsulphonephthalein test described by Silverton, of Sydney, had been combined with it. In Silverton's test one cubic centimetre of the dye was injected intramuscularly. The urine was collected by catheter into a sterile bottle for seventy minutes, and the bottle was marked "1". Urine was collected for a further sixty minutes into a second bottle, which was marked "2". Each specimen was diluted with water to one pint and made alkaline with *liquor potassæ*. Samples were taken in specimen glasses of equal size and held up to the light for comparison of colour. A normal kidney excreted dye much more strongly during the first hour than in the second. The contents of the first glass would be at least a medium magenta colour and that of the second glass a lighter pink. When the renal efficiency was low there might be no colour at all in the contents of the first glass, and merely a pale pink in that of the second.

Harris had recommended the indigo-carmine test, but it seemed to Dr. Davidson that when there was a big post-prostatic pouch the first traces of the dye would not drain out at once; and if water was run in to wash it out, the dye might be over-diluted. Dr. Davidson stated that for some considerable time most of the operations had been carried out under spinal anæsthesia; a heavy solution of "Percaine" had proved satisfactory to him and to Dr. Percival Smith, who had usually administered it. For the previous two or three years Dr. Davidson had endeavoured to carry out the Harris technique, but he had been in the habit of leaving a small self-retaining catheter in the suprapubic wound for five days so that the bladder had not been completely closed. He did not feel that this modification was a disadvantage, and in a large proportion of the cases the tissues had fallen together after the removal of the suprapubic tube, by which time the urethral catheter was sufficient for drainage. As a rule, after two weeks the wounds had

almost healed and natural micturition was taking place. He thought that the essential features of the Harris operation were the positive control of hæmorrhage, the obliteration of dead spaces and the retrigonization of the prostatic cavity; by comparison with these features, complete closure was a secondary consideration. He had carried out bilateral section of the vas at the time of operation. He had used suction drainage, which was laid on in the surgical wards, when some delay had occurred in closing or if a larger tube had to be used for the draining.

Dr. Davidson demonstrated a very large vesical calculus which he had taken from the infected bladder of an obese male patient, whose prostate also had been very large. The patient had been too ill and his bladder too dirty for anything other than a two-stage operation to be considered; but eventually he had become fit to have his prostate removed. It was apparent that the presence of the stone had caused the prostate, which was definitely adenomatous, to become set in tissues resembling concrete in consistency; and the presence of the suprapubic tube had caused a similar condition in the tissues through which he had made the incision. When eventually the gland had been removed, neither the patient nor the surgeon was in a condition for plastic work on the bladder base. Three rolls of gauze were packed fairly lightly and were brought out alongside a medium-sized suprapubic tube. Suction drainage was instituted, and finally the wound had closed up and the patient had micturated normally.

Though Dr. Davidson realized that the value of suction drainage was not unquestioned, he considered that it was essential to keep the patient quite dry; a great saving in dressings and in laundering bed-clothes was thus effected, and when suction was applied properly he did not think that it interfered in any way with the healing of the wounds. With reference to transurethral prostatectomy, Dr. Davidson said that he had obtained the best results in median bar formations and fibrous prostates. Middle-lobe enlargements of moderate degree had responded satisfactorily, though he felt that prostatectomy, in the absence of general contraindications, was the operation of election when well-defined adenomata were present. There were, however, instances in which the major operation seemed to be out of the question, and he thought that generally it was possible to relieve the obstruction by transurethral methods, even when the adenomata were large. It had been his experience, in the few cases of this type he had encountered, that generally it was necessary to repeat the procedure more than once, though possibly, as his experience increased, multiplicity of operations would not be necessary. For the transurethral operation he had used either sacral or low spinal injection of the anæsthetic, and the instrument he had used was Canny Ryall's modification of the McCarthy prostatic electrotome.

Dr. Davidson referred to hæmorrhage as the most worrying factor apart from the many technical difficulties. When the hæmorrhage occurred as a prolonged ooze, clot retention was likely. It was a serious complication, calling for early open operation. It had occurred in one of the patients of the present series and was responsible for death. He had endeavoured to obtain from America a Folly's bag, which was reported to control hæmorrhage satisfactorily. He understood that it acted in much the same way as a Pilcher's bag: it was passed down the urethra and was slightly distended to exert pressure to control the hæmorrhage. Another big worry was sepsis, the control of which largely depended on suitable pre-operative and post-operative treatment.

DR. C. J. O. BROWN congratulated Dr. Davidson on the results he had obtained. At the Alfred and Royal Melbourne Hospitals the mortality rate for prostatectomy operations was about 12%, though at the special hospitals the results were better. Harris's mortality rate for the operation was about 2·3%, and Fryer's was 5%; and as Davidson's rate for suprapubic prostatectomy was so far under 6%, his results were very good. Dr. Brown thought that it would be easier in Ballarat to get the patients admitted to hospital earlier; retention of the urine should indicate admission to hospital and should be treated in hospital; drainage could be established immediately through an indwelling catheter. On the other hand, in town it was necessary to catheterize the patients in the casualty department and to send them home because of the difficulty in obtaining beds for them in the general hospitals. Some of the patients came up daily or several times a day; the treatment was longer and the mortality was higher. Between 80% and 90% of the patients should be subjected to one-stage prostatectomy, though at the Alfred Hospital it was probable that a much lower percentage were fit for it. The mortality rate for suprapubic cystostomy was higher than that for secondary prostatectomy, but one-third of the patients with prostatic enlargement who were subjected to cystostomy did not get beyond this stage. The mortality rate of suprapubic drainage was between 15% and 16%, and among those who survived for the second operation it was 7%.

DR. A. E. COATES also congratulated Dr. Davidson on the excellent series of cases. He agreed with Dr. Brown that the mortality figures were very low. At the Royal Melbourne Hospital the patients were not all suitable for the Harris technique; many were derelict patients and some of them came from the country. The Melbourne general hospitals were in the nature of infirmaries, but this should not deter the surgeons at these hospitals from trying to emulate Dr. Davidson's figures. He had to admit that under general hospital conditions surgeons were often unable personally to attend to the details of the preliminary treatment and the after-treatment with the requisite precision. The onset of pyelonephritis a week or two after operation killed some of the patients who had done quite well in other respects. This type of surgery could be improved in the hands of specialists, but there were so many patients of this type at the general hospitals in Melbourne that each of the surgeons found he was called upon frequently to undertake the work.

A MEETING of the Queensland Branch of the British Medical Association was held at British Medical Association House, Wickham Terrace, Brisbane, on September 3, 1937.

Anæsthetic Accidents.

DR. HORACE W. JOHNSON read a paper in which he discussed anæsthetic accidents.

Dr. Johnson said that when he was first asked to read a paper on anæsthesia at the Branch meeting he was confronted with the difficulty of choosing a suitable subject. The art of giving anæsthetics of any kind was impossible to teach except in the operating theatre. He thought, however, that by dealing with the accidents, both major and minor, which cropped up during the course of anæsthesia, and by giving some hints as to their avoidance, the paper would serve a useful purpose. Dr. Johnson apologized for the apparently elementary nature of some of his remarks, but said that they aimed at the correction of faults which commonly occurred and which might be avoided by the exercise of a little care.

Unexpected events or events occurring without apparent cause during anæsthesia might be most trivial or most serious; even that most serious of all anæsthetic accidents, the death of the patient, might occur. A very detailed classification would therefore be necessary to include every type. Dr. Johnson would not attempt to give every possible accident, but would mention only those of which he had had personal experience. He intended to deal mainly with inhalation methods of anæsthesia. Local and spinal anæsthesia would not be included.

To enable the subject to be dealt with more easily, he had divided accidents into two classes: (*a*) those common to all inhalation methods, (*b*) those peculiar to one particular type of anæsthetic. He dealt first with the accidents common to all inhalation methods. These could be divided into (*a*) those occurring during induction, (*b*) those occurring during maintenance of anæsthesia, (*c*) those occurring during recovery from anæsthesia.

Speaking first of accidents occurring during induction, Dr. Johnson said that their effects or the fact that they had occurred might not be noticeable until later. It was difficult to classify them satisfactorily, so he would deal with them as they had occurred to him.

Accidents arising from mistakes in the anæsthetic agent were fairly common. It was extremely difficult to anæsthetize a patient with a bottle of distilled water, or even with methylated spirits; yet he had seen both of these used when the anæsthetist thought he was using ether.

It seemed impossible to confuse chloroform with ether, yet this had been responsible for quite a number of fatalities. After some hours' work in a hot operating theatre it was quite easy for a tired anæsthetist to smell the bottles and even then not be quite sure which contained ether and which chloroform. If any doubt existed, it was best to pour some into the palm of the hand and taste it—the sweet burning taste of chloroform was unmistakable. Chloroform and ether mixtures were sometimes confused with simple ether. The anæsthetist's rule should be always to fill his own bottles and ether tank. Only by this means could mistakes be lessened.

Dr. Johnson then referred to self-injury from struggles during induction. The commonest of these was injury from rings or jewellery worn by the patient. It was wise to remove all such articles before induction was commenced. Injury to the body from struggles during induction was a fairly common accident at this stage. Bruises at the back of the heel from kicking onto a hard table were common in children; this could be avoided by efficient restraint by an experienced assistant.

The essential from the anæsthetist's point of view was that the mask over the face must not be touched by the patient. In avoiding this, fingers or wrists were often hurt by the assistant, with consequent complaint from the patient afterwards. The essential for good induction in an adult patient was a comfortable position. Absence of any attempt at restraint would also make for a calmer mind and thus for more pleasant anæsthesia. In adults he found that the easiest method was to have the fingers locked together across the chest. Any movement or muscular spasm of the hands then tended only to tighten the patient's grip and to restrain him more. This tip was worth remembering. It was much simpler and more satisfactory in every way than the usual method of hands by the side.

With children, if the four fingers, but not the thumb, of each hand were held by the assistant, the mask could never be pulled off by the patient, however strong he might be and whatever arm movements he might make. By this means one avoided the unpleasant experience of having the mask snatched away when the induction was only half completed.

When the patient lifted his head and shoulders from the table, it was better to follow the patient up with the mask rather than to attempt to hold his shoulders and head still—a difficult procedure in a strong man. If this was impossible, it was much easier to keep head and shoulders on the table by lifting the legs rather than by forcing the upper part of the trunk back onto the table. Of straps Dr. Johnson did not approve, except in very exceptional circumstances, as they tended to set up a subconscious antagonism in the patient, reflected in his actions as soon as the higher cortical centres were depressed.

Burns to the face from the anæsthetic agent were fortunately rarer since the use of chloroform had decreased. It was advantageous to smear the face well with "Vaseline" if chloroform was being used as the sole anæsthetic.

Injuries to the eyes were fairly common, yet they were practically all avoidable. A lot were caused by the anæsthetist trying corneal and conjunctival reflexes with the tip of the finger and thus causing small corneal abrasions. The conjunctiva should rarely and the cornea should never be touched with the finger to determine the depth of anæsthesia. If it was necessary to try the corneal reflex, this could be done quite effectively and with a minimum of trauma by gently blowing on the opened eye. Eyes should be opened by pulling on the top lid. Injury was often done to the eye by ether dropping into it from a saturated mask. The instillation of castor oil drops was one of the recommended means of counteracting eye injury; its use was probably only one of the superstitions that survived in medicine.

Whether castor oil was of use as a means of avoiding "anæsthetic eyes" or not, it was certainly of some use in making the eyelids adhere and remain closed, and for this reason Dr. Johnson often used it. For goitre patients or for the occasional patient who persisted in keeping his eyes widely staring during anæsthesia, he found it best to stick the lids together with small pieces of sticking plaster or "Elastoplast". This was easily put on, the eyes were easily opened to study eye reflexes, and it was easily cleaned off at the end of operation. This sticking of lids together certainly stopped the horrible sight of a cornea dried to the point of desiccation, with the imprint of a gauze mask patterned across it in lattice-work design.

Accidents to the lips were common. A patient in spasm would often bite through a lip unless care was taken to see that the lips were not caught between the teeth. Apropos of lips, after dental or jaw operations a careful cleansing, massage and smearing of the lips with "Vaseline" made for a less trying convalescence.

A survey of the mouth was necessary before all induction. Good fitting full dentures were usually better left in for all anæsthetics; in gas anæsthesia their presence was almost an essential in order to obtain an air-tight system. Ill-fitting plates and partial plates were better removed. Loose teeth should be noted, so that any pressure on them could be avoided. With quite small children inspection of the mouth was just as important prior to anæsthesia as it was to ask an adult about dentures. Dr. Johnson remembered giving an anæsthetic to a child for a cleft palate operation. When the patient was wheeled into the theatre and the mouth was gagged open at the commencement of operation, he had been, to say the least of it, surprised to see the surgeon remove from the patient's fauces part of a "Mintie" wrapping about an inch square. Foreign bodies of this type could quite easily have serious consequences if inhaled into the larynx or trachea.

All of these accidents of induction had been of a minor nature. He now came to four of very much greater importance—respiratory spasm, obstructed airway, respiratory failure and primary cardiac failure.

Respiratory spasm occurred frequently during induction. Some degree of spasm was almost always associated with ethyl chloride induction. Spasm and breath-holding could as a rule be considerably lessened by careful grading of the concentration of anæsthetic vapour. Sometimes the spasm was a spasm of lips only, and it was always worth while slipping the hand under the mask and holding the lips apart with the fingers to see whether the procedure would improve matters. Once laryngeal spasm had occurred and remained for some time, pouring on of anæsthetic would not do anything to hasten anæsthesia. One often saw patients after twenty or thirty minutes of anæsthesia still with a laryngeal spasm, small pupils, moving eyes and clenched jaws while the mask was saturated with ether. If a supply of "Carbogen" was available, a few whiffs of this would often act like magic—the respirations deepened, the glottis relaxed and anæsthesia soon supervened. If no "Carbogen" was available, it was usually quicker to take the mask off and let the patient come right out before recommencing a better graded anæsthesia.

"Carbogen" was also of great use in the treatment of a patient who had had a little too much premedication. In this case the respirations were often cut down to such an extent that not enough anæsthetic vapour could be inhaled to get the patient under. "Carbogen" here soon put matters right. Two or three breaths of "Carbogen" were also of great use just before the passage of an endotracheal tube, as they widened the glottis and allowed easier intubation.

Respiratory spasm was often caused by an attempt to insert an airway before the anæsthesia was deep enough for the patient to stand it.

Obstructed airway was a common accident during late induction. The forcing of the mouth open with a gag at this stage and pulling on the tongue were rarely necessary and often harmful. Obstructed airway was common in patients whose dentures had been removed; in these circumstances it was caused by the tongue falling backwards against the roof of the mouth, and often it could be overcome only by the introduction of an artificial airway.

Obstruction from vomiting of food was a common anæsthetic accident, especially in children who had had meals too large or too late before anæsthesia. To adults a stomach wash-out could often be given, but with a sick child the upset was often too great when there were other simpler means available. The method which Dr. Johnson used to get rid of stomach contents, when their presence was suspected in children, was to place the child on its side and to induce anæsthesia with ethyl chloride followed by ether until the point was reached at which the swallowing reflex was abolished. This was easily determined by holding the fingers under the mask. The mouth was now opened with a gag and after a few seconds a pharyngeal reflex returned to the point at which stimulation with a tongue depressor induced vomiting. By this means the stomach emptied itself without distressing the semi-conscious patient.

Obstruction from bronchial secretion was a very common occurrence in ether anæsthesia. Atropine in sufficient dosage was essential for its control. If the dose had not been enough to control secretion, obstruction to the airway occurred and asphyxia with cyanosis soon followed. Dr. Johnson found that the mopping out of the secretion was practically useless. The mucus reformed almost as soon as the mouth was mopped dry. It was much easier to tilt the head on one side, put a small pillow under the shoulders and let the bronchial secretion gather in the side of the cheek, whence it ran out from the corner of the mouth. An extra dose of atropine should be given immediately. If a sucker was handy, he always used it to remove secretion until the atropine had had time to act.

Respiratory failure was an accident caused usually by overdose. When it occurred there was probably no need to worry if the pulse was good and the colour one of cyanosis only. If breathing did not start after five or six seconds, artificial respiration should be commenced. Often the mistake was made that respiration had ceased when this was not the case. A small wisp of cotton wool held in front of the mouth would often show that very shallow respirations were still taking place, in which case removal of the mask only would often suffice and the operation could be continued without interruption.

It was important, if artificial respiration was to be given, that the operator should have free access to the lower ribs. Dr. Johnson had recently had experience of a case in which the surgeon was putting on a pelvic plaster. The child stopped breathing from some unknown cause. Dr. Johnson did not think that it was overdosage. Attempts at artificial respiration were made with the plaster still *in situ*, but they were unavailing and quite useless. The plaster was removed totally in a few seconds with a pair of shears, and a few pumps on the chest soon restored normal respiration.

Referring to obstructed airways, Dr. Johnson said that one sound piece of advice was always to count the number of swabs put into the mouth by the dentist. He well remembered when he was anæsthetist at a hospital in London, it was the custom on dental extraction morning for himself and a student to work at adjoining dental chairs giving gas for extractions. He was responsible for the anæsthetics given in the room. One day he had just finished his anæsthetic when he saw the patient in the adjoining chair being lowered to the floor. She had stopped breathing and was absolutely cyanosed, the pulse still feebly flickering. Artificial respiration was being done, the tongue was pulled out, "Carbogen" was blowing all over the pharynx, but no improvement in colour was taking place. He asked the dentist whether the swab from the back of the mouth had come out. The dentist assured him that it had, and even produced it as evidence. Not satisfied, he pushed his finger as far down the patient's throat as he could and dug out a small swab lying over the glottis. Artificial respiration soon restored the patient. Evidently two swabs had been picked up together and used by mistake, and only one of them had been removed. This had happened to him only once, but since then Dr. Johnson always counted the swabs going into a mouth.

If breathing did not recommence after artificial respiration and the pulse showed signs of weakening, then the treatment of the condition became the same as that of primary cardiac failure.

Primary cardiac failure was the most serious of accidents which could occur during induction; it most commonly occurred during light anæsthesia with chloroform.

Primary cardiac failure was not a common accident, but it was such a serious accident that it was well to have some routine standard method of treatment. This had been well summarized by Langton Hewer in the last edition of his "Recent Advances in Anæsthesia".

Whatever might be the stage of the operation, it should be suspended at once and the patient should be tilted into the steep Trendelenburg position. There was no doubt that immediate inversion had frequently led to recovery.

The lungs should slowly and rhythmically be inflated with a 5% carbon dioxide–95% oxygen mixture, or, failing this, with pure oxygen. If no "Carbogen" outfit was available, then efficient artificial respiration should be instituted. The "respiratory pump" effect of the forced breathing would aid a feeble circulation, provided that the circulation was still in existence. At the end of two or three minutes the situation had to be reviewed. If there was no improvement in colour or contraction of the pupils and if no pulse or heart beat could be detected, it was obvious that no efficient circulation was present, and immediate steps should be taken to restore it.

Injection of ten minims of adrenaline or one cubic centimetre of "Coramine" into the heart should be carried out. The injection was preferably given into the third right intercostal space, close to the sternum downwards and towards the mid-line to a depth of three and a half inches. Dr. Johnson was thankful that he had never had to do this, but it was necessary to be prepared to do so if the occasion arose.

If cardiac puncture had failed to elicit a response, massage of the heart through an abdominal incision should be attempted without delay. There was no doubt that this had in some cases restored life.

Dr. Johnson said that up to that point he had been speaking of the accidents occurring during induction. He now came to the next group of accidents, namely, those occurring during the course of the anæsthetic. Many of the accidents given under this heading occurred, of course, also under the other stages.

With regard to nerve injuries, injury to the musculospiral nerve was fairly common; so also was injury to the brachial plexus due to stretching of the arms above the head. Other nerves occasionally injured were the circumflex and the long thoracic nerve.

Referring to accidents arising from the use of airways, Dr. Johnson said that he used airways practically as a routine measure. It saved work holding the jaw forward, and in many cases kept the patient a much better colour with less trouble. Airways should never be inserted unless they could be inserted easily. No forcing open of jaws should be necessary. In edentulous patients they were often a necessity, as the base of the tongue fell backward and obstructed the airway. Insertion could do a lot of damage to the back of the throat. To guard against this, the airway should be wetted or greased; it then slipped in more easily and less damage was done.

In discussing accidents arising from the use of gags, Dr. Johnson said that teeth could easily be injured by the careless use of a gag. Gags with pieces of rubber at the end of them were a possible source of trouble. He had heard of at least one case in Brisbane in which the piece of rubber on the end of the gag was lost during the operation, to be retrieved at a later date from a bronchus by the ear, nose and throat surgeon.

Pressure from the mask in gas anæsthesia was fairly common. Deflation of the rubber cushion occurred and in an attempt to make the system airtight the mask might be too tightly pressed on the face, resulting in bruising and skinning of the nose. This took a bit of explaining to the patient next day.

Accidents due to faults of the operating table itself occasionally occurred. He had seen two patients fall completely to the floor during the course of an operation. Obviously experience of the working of the operating table was an essential for the average anæsthetist. One of the grudges he had against being an anæsthetist was that he was compelled to be too handy. Jobs such as fixing the table, adjusting the light, lifting the patient and boiling up odd instruments that dropped, putting on tourniquets *et cetera*, all fell to his lot and were perhaps part of his work; but wiping the surgeon's nose, adjusting the sister's cap and retrieving slipping clothes were, he thought, a little outside the anæsthetist's sphere.

One pitfall to avoid was too much pressure behind the jaw. He had had several complaints from patients after operation of pain behind the angle of the jaw. He had even seen one case of severe bruising.

Ether convulsions were one of the major accidents occurring during the middle stage of anæsthesia. Such a lot had been published about this subject recently that he did not intend to deal with it. In his experience ether convulsions had always occurred during deep anæsthesia in a toxic patient when oxygen was being given as well as ether. Probably in these cases the anæsthesia was much deeper than the anæsthetist realized, owing to the fact that the oxygen prevented cyanosis, which was the usual danger signal with other modes of anæsthesia.

Dr. Johnson then dealt with accidents occurring during the recovery stage of anæsthesia. There were only two common accidents.

Obstruction of the airways from vomitus was probably the most common accident during this stage. When possible, the anæsthetist should always accompany the patient back to the ward and see that a good airway was established before handing him over to the sister's care. It was better, if the nature of the operation allowed it, to turn the patient on his side during the recovery stage, and to change this position every hour or so. By this means a lot of post-operative pain in the back would be avoided. If this turning on one side was impossible, at least the head should be turned to one side. As Robson in his article on anæsthesia in a recent number of the *American Journal of Surgery* stated, many a patient was figuratively and literally looking towards heaven, when he was lying in an anæsthetic state with his face directed upward.

When a Boyle-Davis gag was being used, it was advisable to remind the surgeon to remove the blood clot from behind the soft palate with a sucker before the conclusion of the operation. This was not always done, and as the clot was often quite a large one, it could easily slip over the glottis and cause complete respiratory obstruction.

Dr. Johnson then turned his attention to accidents peculiar to one type of anæsthetic.

He spoke first of endotracheal anæsthesia, as it was the most important.

Accidents to teeth were quite common. It was preferable to stick "Z.O." plaster along the teeth before endoscopy to prevent chipping of the enamel. With regard to endoscopy, a good assistant was an extremely important aid in the passage of an intratracheal tube. A poor or inexperienced assistant was a hindrance. It was extremely easy to dislodge teeth during endoscopy. Loose teeth were almost sure to go if pressed upon by a laryngoscope.

Injury to the posterior pharyngeal wall from the blade of the endoscope sometimes occurred. The manipulation was made easier if the blade was smeared with paraffin beforehand.

It was essential to see that the tube was in the larynx and not in the œsophagus. Dr. Johnson had had the experience twice of seeing an abdominal tumour rising slowly from among the drapings.

It was imperative to turn the machine on and to sample the delivery of vapour before connecting it to the intratracheal tube. Never should the connexion be made first and the machine switched on afterwards, as the machine was sometimes connected incorrectly through no fault of the anæsthetist and the anæsthetic agent, still in its liquid form, might be blown directly into the bronchi with disastrous results. The patient usually died immediately from shock.

A manometer or some substitute for a manometer was essential in all endotracheal work. Dr. Johnson used a two-way safety by-pass, as he found that the mercury in the ordinary manometer was too easily spilled when being carried around; this safety let-off acted as a valve for the escape of any excess pressure, and also allowed a keen following of respiratory movement. A piece of wool balanced or, better, stuck on with a piece of the ever useful "Z.O." gave one a visual as well as an auditory guide to the respiratory movements. The importance of the manometer or of the safety valve could not be too strongly stressed. He had seen two cases of surgical emphysema following endotracheal anæsthesia.

Accidents with intravenous anæsthetics were few in Dr. Johnson's experience, probably because of the relatively small number of these that he had given. The usual intravenous anæsthetics were "Evipan Sodium" and "Pentothal Sodium", both barbiturates. Sore arms through the vein not being entered correctly were not common with "Evipan Sodium", though they were said to be more common with "Pentothal Sodium".

Everyone should know that it was very unwise to supplement barbiturates given orally by barbiturates given intravenously, as the anæsthesia from oral administration might be slow through delayed absorption from the stomach and the cumulative effect of the combined oral and intravenous doses might be very dangerous.

Amnesia might be one of the accidents or, rather, sequelæ of "Evipan" injection. It was not wise to allow a patient to go from the surgery or dentist's rooms unattended, however rational he might seem. Dr. Johnson had seen a patient talk and act quite rationally for some four hours after an injection of "Evipan Sodium" and yet next day have absolutely no recollection of those hours or of how he had spent them.

Spasm of the part that was being handled was one of the interesting accompaniments of "Evipan" anæsthesia that he had often noticed. For this reason it sometimes failed completely when manipulation of the part was required. He had also noticed that patients who were having teeth extracted or fractured mandibles manipulated were prone to laryngeal spasm. A few moments' cessation of all manipulation was enough to cause a relaxation of the glottis and then the operation might be continued.

The most formidable accident, of course, with the barbiturates was respiratory failure due to overdose, though he had never seen a case of this. The antidote recommended varied. One author had recently suggested 0·5 gramme of caffeine with 0·5 gramme of sodium benzoate given intravenously. Malony, writing in the *American Journal of Surgery* of December, 1936, stated that two cubic centimetres of a 0·3% solution of picrotoxin should be injected intramuscularly into the deltoid before any "Evipan" anæsthetic. By this means, the author claimed, all respiratory depression was abolished. "Coramine" and "Lobeleine" also had their uses as correctives for overdosage.

Accidents following the use of paraldehyde and "Avertin" were usually those of underdose or overdosage. Dr. Johnson had not used these drugs to any great extent; but had seen delirium and excitement with both of them from (he thought) underdosage. Colitis was also stated to be a common sequel to both of them, but he had never seen a case in which colitis could be definitely attributed to the anæsthetic agent.

Dr. Johnson had seen ether given by the rectum on only two occasions. In one case it was given for a plastic facial operation. Not only was the operation lasting three hours quite uneventful with perfect anæsthesia, but the patient

was still quietly snoring when the first dressing was done thirty-six hours later.

In the use of machine anæsthetics the most important factor was the explosion risk. The anæsthetist should remember with any machine that this was always liable to occur, especially in the type in which the motor was near the ether tank. Dr. Johnson had seen three such fires with ether machines. One was a serious fire in which the patient was badly burnt. In each case the machine was of the type in which the motor was close to the ether tank. Worn parts, leaky taps and faulty switches did the rest. The machine became old, joints were loosened, leaks developed and a sparking machine soon detected an ether leak. All machines should be regularly overhauled.

A safety bottle should always be put in circuit between the machine and the patient, though these were not always infallible. In a darkened theatre, while giving ether recently by the intrapharyngeal method for a radical antrum operation, he had noticed a sudden straightening of the tube that led to the patient, just as a hose straightened out when the water tap was first turned on. When he hurriedly pinched the tube and disconnected it, liquid ether ether poured out of the end of the tube. What had happened was that the ether tank had been tilted and liquid ether had poured over into the safety bottle, filled it, and then started off on its way to the patient's pharynx. The filling of the safety bottle had not been noticed as the theatre was in darkness.

The use of surgical diathermy or cautery or the presence of a naked light was an absolute contraindication to the use of ether. When either diathermy or cautery was being used it was a wise rule to put the ether outside the operating theatre before the operation began.

Choloroform was the best anæsthetic to use for operations such as diathermy of a malignant tonsil. A word of warning was necessary about the use of oxygen in such cases. In some places it was the custom to use oxygen under pressure as a means of blowing chloroform or ether into the patient. Now oxygen greatly increased the risks of conflagration. Certain mixtures of chloroform and oxygen formed an explosive mixture, as he had once discovered. While a tonsillar growth was being treated by diathermy, there was a sudden pop, everyone leapt in the air, and the surgeon, who by the way had an exophthalmic goitre, made for the door, but returned in time to help extinguish some cotton wool in the patient's nostrils that had caught alight. Apart from a few superficial burns inside the mouth of the patient, there were no serious after-effects.

Ethylene, as was well known, was most explosive in certain concentrations. Static electricity was given as the cause of some explosions, and for this reason some anæsthetists always earthed their machines and the operation table when ethylene was being used. "Cyclopropane", which was now commonly used in America, and which was rapidly gaining in popularity in Australia, was less explosive, but still could not be used in the presence of naked flames.

Dr. Johnson said that this concluded the rather ragged list of anæsthetic accidents which it had been his lot to encounter. Many of importance had been overlooked; many trivial accidents had been included; but they were all of some importance when the well-being of the patient was being considered. So that they might be avoided, he wished to give this advice to all anæsthetists, whatever their experience: "Be alert, be aware, but, above all, keep awake." They should remember that now classical remark made by an annoyed surgeon to a nodding anæsthetist: "Really, doctor, I think that if the patient can keep awake, the anæsthetist should."

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Turnbull, Gordon McLeod, M.B., B.S., 1937 (Univ. Adelaide), Adelaide Hospital, Adelaide.

Hayes, Desmond Thomas Matthew, M.B., B.S., 1936 (Univ. Melbourne), Adelaide Children's Hospital, Adelaide.

Smith, Gavin Viner, M.B., B.S., 1937 (Univ. Adelaide), 45, Marlborough Street, St. Peters.

Greenlees, Rollo, M.B., B.S., 1937 (Univ. Adelaide), Adelaide Hospital, Adelaide.

Davis, Hubert Garth, M.B., B.S., 1937 (Univ. Adelaide), Adelaide Hospital, Adelaide.

The undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

MacIntosh, Alexander Mackay, M.B., B.S., 1938 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Alderdice, Alexander Arthur, M.B., B.S., 1936 (Univ. Sydney), c.o. English, Scottish and Australian Bank, 5, Gracechurch Street, London, E.C.3 (temporary).

Atkins, Donald Stewart, M.B., B.S., 1937 (Univ. Sydney), Royal South Sydney Hospital, Zetland.

Beckett, Charles Edward Halley, M.B., 1936 (Univ. Sydney), 283, Cleveland Street, Redfern.

Blaxland, Gregory McLeod, M.B., B.S., 1937 (Univ. Sydney), 15, Chertsey Street, Merrylands.

Flynn, Gregory Stephen, M.B., B.S., 1935 (Univ. Sydney), 135, Macquarie Street, Sydney.

Frost, Alan Dudley Joseph, M.B., B.S., 1936 (Univ. Sydney), 16, Birriga Road, Bellevue Hill.

Harrington, Charles Frederick, M.B., B.S., 1938 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Leleu, Charles John Newhill, M.B., B.S., 1936 (Univ. Sydney), Kembla Street, Wollongong.

Loewenthal, John Isaacs, M.B., B.S., 1938 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Mathieson, John Bryan, M.B., B.S., 1937 (Univ. Sydney), Sydney Hospital, Sydney.

Perkins, Richard Basil, M.B., B.S., 1936 (Univ. Sydney), 12, Parkview Road, Manly.

Smith, Gordon Clive, M.B., B.S., 1935 (Univ. Sydney), 15, Blenheim Road, Lindfield.

Speirs, Robert Bradley, M.B., B.S., 1938 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Correspondence.

ANTERIOR POLIOMYELITIS.

SIR: In the excellent report of the Queensland Commission Battens' zero position for rest is advocated. T. S. Ellis ("The Human Foot", published fifty years ago) pointed out that the optimum rest for muscles acting on a joint was midway between its limits of movement.

Reeducation of muscles, so-called, was introduced by Ling, of Sweden, over a hundred years ago, to improve the physical fitness of the nation, and should not in my opinion be applied to muscles whose nerve supply is damaged by an inflammatory lesion while still active.

Why diathermy is banned by those controlling the epidemic in Victoria passes my understanding. Professor Bordier, of Lyon, has used it for twenty-five years, and many of his colleagues are quoted in the sixth edition of his book on diathermy as enthusiastic.

At the first "Short Wave Conference" held at Vienna in July, 1937, and reported in *The Lancet*, August 14 and September 17, 1937, several medical men spoke in favour of its use in anterior poliomyelitis. No one seems to have used it in old-standing cases except myself. Bordier's oldest case was eighteen months after onset, with complete recovery. My best result to date was aged twenty years—fifteen years after onset.

Nearly every case shows some improvement; some show none; and quite a good percentage show great improvement.

Heliotherapy is another agent sadly neglected in Victoria.

Rest is a difficult problem. It must be used for a sufficient period—weeks if not months in certain cases. But prolonged periods up to five years is wicked—just as wicked as it is to deny cases of two years after onset chances of recovery free of charge.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,
Melbourne,
February 9, 1938.

"AN EMBARRASSING MISHAP."

SIR: A rather embarrassing mishap in an abdominal operation, that I have not seen mentioned in the literature, was described to me by a middle-aged woman whom I was examining. She had a well-healed abdominal scar. On inquiring as to what operation had been performed, she told me that the "womb had been stuck to the bladder". From that I gather that some uterine displacement had been remedied. She enlarged on the severity of the operation, and assured me that when she was "opened up all my insides fell out". Now this must have been very disconcerting to the surgeon. But I think it speaks volumes to his skill, as it appeared to me that her somewhat capacious abdomen still contains a good many organs, so that apparently they were all, or nearly all, satisfactorily replaced.

Yours, etc.,

"F.A.R."

January 29, 1938.

THE COSMETIC VALUE OF THE UMBILICUS.

SIR: Apropos of the letter of Dr. W. John Close about the cosmetic value of the navel, I think the following incident which happened to me might be interesting.

One evening I was called to a lock-up in Queensland to see a man arrested in the street on suspicion of being of unsound mind. There was no history available. I had a talk with him, and just as I would think I had some delusion on which to base a certificate of insanity, each time the patient would correct the delusion. I was fairly satisfied in my mind of the insanity of the patient, but it was difficult to find exact points to set down on a certificate of insanity, so I ordered food and tea for him, adjourning further examination till the following morning.

As I was leaving the cell, the man called out: "Are you a doctor?" The lock-up keeper, a big burly man, said: "Of course he is." I asked the man what he wanted. He asked me to look at his leg, and I told him to show it to me, which he proceeded to do by rolling up his trouser, but in doing so at the same time tried to hide something beneath his sock above his boot. I asked him what it was, but he declined to show it to me, and then the lock-up keeper, in stentorian tones, told him at once to show it to the doctor. The man then disclosed a piece of string, loosely tied around his leg, and on the string were a number of buttons, of from about 12 to 20 in number, of different kinds.

I asked him what they were for. He looked at me with a cunning leer and smile, and said: "Don't yer know?" Then, pointing to the region of his umbilicus, said: "If you lose your body button, they'll say you are a button short and put you into an asylum, so I always carry these and if one of my friends is a button short I can give him one."

A little more talk and I was able surely to fill in the required certificate.

Yours, etc.,

F. H. VIVIAN VOSS, F.R.C.S. (Eng.).

106, Bellevue Road,
Bellevue Hill,
Sydney.
February 14, 1938.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

AT a recent meeting of the Senate of the University of Sydney the following appointments were made.

Professor J. C. Windeyer, who was appointed to the part-time Professorship in Obstetrics in 1925, has now been appointed as full-time Professor in the Department of Obstetrics as from January 1 last. This will enable Professor Windeyer to devote more time to his own research as well as supervising important research work that is being carried on in his department at the present time. He will hold the position for three years.

Three members of the Professorial Board who have held the position of Associate Professor, have been promoted to the full grade of Professor. They are: Professor E. G. Waterhouse, who is now Professor of German; Professor H. Priestley, now Professor of Biochemistry in the Faculty of Medicine; Professor F. A. Eastaugh, now Professor of Engineering, Technology and Metallurgy. All three have rendered yeoman service to the university for some years, and their promotion has met with universal approval in academic circles.

Dr. S. A. Smith and Dr. H. R. G. Poate have been appointed by the Senate to the positions of Lecturer in Post-Graduate Medicine and Lecturer in Post-Graduate Surgery, respectively, at the Prince Henry Hospital. The Senate understands that these two gentlemen will be appointed directors of the respective units of the hospital. The positions will be half-time appointments for the present. It is proposed to open the Post-Graduate School at the Prince Henry Hospital on the afternoon of Tuesday, April 5, 1938.

Dr. A. Bolliger has been appointed as full-time Director of the Department of Urology in the Department of Surgery.

Benefaction: The University has been advised by the Perpetual Trustee Company, executors of the estate of the late Herbert Johnson, who was a director of David Jones Limited, that he had left his estate of approximately £17,000 to the University for the establishment of travelling scholarships for necessitous students. The executors have informed the University authorities that the money will not be available for some time, as other beneficiaries will first benefit of the income from the estate.

NOTICE.

DR. H. FLECKER, of Cairns, is anxious to secure small fœtuses, of between thirty and sixty-two millimetres in length, measured in the fresh condition from crown to rump. These are being sought for radiological research. If specially desired, they will be returned to the sender immediately after examination.

Books Received.

SPEECH TRAINING FOR CASES OF CLEFT PALATE, by M. C. Oldfield, M.Ch., F.R.C.S.; 1938. London: H. K. Lewis and Company Limited. Imperial 8vo, pp. 23, with 38 illustrations. Price: 4s. 6d. net.

MEA CULPA AND THE LIFE AND WORK OF SEMMELWEIS, by Louis-Ferdinand Céline, translated by R. A. Parker; 1937. London: George Allen and Unwin Limited. Crown 8vo, pp. 175. Price: 5s. net.

THE BASIS OF TISSUE EVOLUTION AND PATHOGENESIS, by A. A. Gray, M.D., F.R.S.E.; 1937. Glasgow, Jackson, Son and Company. Demy 8vo, pp. 101, with illustrations. Price: 7s. 6d. net.

Diary for the Month.

FEB. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 23.—Victorian Branch, B.M.A.: Council.
FEB. 24.—South Australian Branch, B.M.A.: Branch.
FEB. 25.—Queensland Branch, B.M.A.: Council.
MAR. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 2.—Western Australian Branch, B.M.A.: Council.
MAR. 2.—Victorian Branch, B.M.A.: Branch.
MAR. 3.—South Australian Branch, B.M.A.: Council.
MAR. 4.—Queensland Branch, B.M.A.: Branch.
MAR. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAR. 11.—Queensland Branch, B.M.A.: Council.
MAR. 15.—New South Wales Branch, B.M.A.: Ethics Committee and Medical Politics Committee.
MAR. 16.—Western Australian Branch, B.M.A.: Branch.
MAR. 22.—New South Wales Branch, B.M.A.: Council (Quarterly).
MAR. 23.—Victorian Branch, B.M.A.: Council.
MAR. 24.—South Australian Branch, B.M.A.: Branch.
MAR. 24.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 25.—Queensland Branch, B.M.A.: Council.
MAR. 29.—New South Wales Branch, B.M.A.: Council (Election of Officers and Standing Committees).

Medical Appointments.

Dr. H. Rogerson has been appointed Superintendent of the Mental Hospital at Kew, Victoria.

* * *

Dr. J. T. Hollow has been appointed Superintendent of the Mental Hospital at Ararat, Victoria.

* * *

Dr. L. T. G. Geraghty has been appointed Government Medical Officer at Gatton, Queensland.

* * *

Dr. D. G. Croll has been appointed a member of the Medical Board of Queensland, according to the provisions of *The Medical Acts*, 1925 *to* 1935, of Queensland.

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Dr. F. O. B. Wilkinson has been appointed Government Medical Officer at Blayney, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii to xx.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Medical Officer.
DOWERIN HOSPITAL BOARD, DOWERIN, WESTERN AUSTRALIA: Medical Practitioner.
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.
THE UNIVERSITY OF MELBOURNE, VICTORIA: Chair of Anatomy.
TAMWORTH BASE HOSPITAL, TAMWORTH, NEW SOUTH WALES: Resident Medical Officer.
WINTON HOSPITAL, WINTON, QUEENSLAND: Medical Superintendent.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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